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MATERNITY MORTALITY IN BOSTON FOR THE YEARS 1933, 1934 AND 1935*

A Study Conducted by the Obstetrical Society of Boston and the Boston Department of Health

IN 1933 the Obstetrical Society of Boston appointed a committee† to make a study of the maternal deaths in Boston for the years 1933, 1934 and 1935. This committee consisted of nine physicians. It was arranged with the Boston Department of Health, through the courtesy of Dr. Wilinsky, to have copies of the certificates of all maternal deaths sent to the committee, on schedules similar to those that had been used in the study of maternal mortality in fifteen states made by the Federal Government and in the New York and Philadelphia studies. These deaths were then assigned to the members of the committee to investigate. It is the analysis of these maternal deaths that we report to you tonight.

The study includes all deaths that were found in which it was stated that the woman was pregnant. It includes more deaths than are reported in the official figures. In the official figures for the City of Boston during 1933 there were 104 deaths, and in 1934 and 1935 there were 87 deaths each year. We have found 23 additional deaths in 1933, 10 in 1934 and 7 in 1935. It is true that some of these additional deaths cannot be regarded as from causes related to pregnancy or childbirth, but we have felt that any death of a woman occurring when pregnancy was present should be included in the total figure, and in the final analysis the death should be put into the proper classification. This of course would include criminal abortions and any obscure death that might not be connected with the obstetric care of the patient, but which to give a proper picture should be included. We have analyzed the causes of 318 deaths. Of these, 284 can be classified under the ten rubrics of the International List of Causes of Death that relate to diseases of pregnancy, childbirth and the puerperal state. The remainder we have classified separately, and they will be taken up later.

*This report was presented at a combined meeting of the Suffolk District Medical Society with the Obstetrical Society of Boston on Wednesday evening, October 28, by Dr. DeNormandie.

†The members of the committee are as follows: Dr. R. L. DeNormandie, Chairman, Dr. Joseph P. Cohen, Dr. John Rock, Dr. Frederick J. Lynch, Dr. R. S. Titus, Dr. D. J. Bristol, Jr., Dr. Louis Phaneuf, Dr. James C. Janney and Dr. A. K. Paine.

Twenty-one of these deaths occurred at home, 1 in a doctor's office, and the remainder died in hospitals. Thirty different hospitals reported deaths. Of the private cases, there were 80 physicians that signed death certificates. Three of these had 3 deaths, 11 had 2 and the remainder had 1 each.

Let us take up one by one the ten classifications according to the International List. First, rubric 140, "Abortion with septic conditions," contains 36 deaths (fig. 1). Twenty-eight of

140. ABORTION WITH SEPTIC CONDITIONS

	No.	Married	Single	No operation	Operation	Medical examiner	Physician
1933	9	6	3	7	2	9	0
1934	11	9	2	2	9	8	3
1935	16	15	1	12	4	11	5
Total	36	30	6	21	15	28	8

FIGURE 1.

these certificates were signed by the medical examiner, which was to be expected, for all of these were probably induced abortions. It is not known whether they were criminal, but is a fair supposition that they were all either self-induced or criminal abortions, for it is rare to have an early spontaneous abortion die from sepsis. Of these 36 women, it is interesting to note that only 6 were unmarried. Twenty-one of them died without any operative interference whatsoever. The other 15 had some operation. Usually it was a dilatation and curettage or a finger curettage. But we call attention to the fact that the operation was done when the patients had temperatures of 100° F. or over. One of these cases had a dilatation and curettage and the uterus was perforated. Ten hours later she showed signs of peritonitis, and the abdomen was opened and drained. In a second case there was an attempt to replace an incarcerated uterus, and the uterus was perforated. The perforation was recognized, and the abdomen

promptly opened and a hysterectomy done. Another patient was desperately sick with pernicious vomiting, refused an early operation; later a dilatation and curettage was done, but she died of sepsis.

Little need be said about the preventability of these deaths. The majority had no operation. They came to the hospital septic, as a result, in all probability, of what they had done themselves or had had done. The major problem here is a sociologic one, and the medical profession can only do its best in trying to save these patients. The two cases in which the uterus was perforated should not have occurred. It was careless operating.

Under rubric 141, "Abortion without mention of septic conditions," there were only 3 deaths. One was caused by an attempt to inject a quart of turpentine into the vagina and uterus. The second went into collapse suddenly after a dilatation and curettage, having bled profusely before this was performed. The third case had also had marked bleeding and was curetted. Both of these might have been saved had they been promptly transfused.

Under 142a, "Ectopic gestation with septic conditions specified," there were 5 deaths. One patient was not operated on because of her very serious condition and the correct diagnosis was made only at autopsy. In 1 there was a delay by the local physician of two days after the onset of symptoms before she was transferred to a hospital. A third case was diagnosed promptly and operated on promptly, an autotransfusion was done, but she died in spite of operation. One full-term abdominal pregnancy was operated on but died from sepsis. In the final case in this rubric the diagnosis was promptly made, and at operation the ruptured tube was removed and a resection of the ovary, an appendectomy and a suspension of the uterus were done.

We feel that multiple operations and appendectomy in the presence of blood in the abdomen are definitely contraindicated. The other case that might have been prevented was that in which there was a definite delay in diagnosis and in sending the patient to the hospital.

Under 142b, "Ectopic gestation without mention of septic conditions," there were 7 cases. One was not operated upon. She was sent into the hospital four and a half hours after onset *in extremis*. In 3 there was a delay of from several days to three weeks before being sent to the hospital. These cases were operated upon promptly and transfused, but failed to rally. The fifth case pointed to an appendix, and she was operated on for this condition and an acutely inflamed appendix was removed. No blood was seen in the abdomen. Two days later she had sudden sharp pain and was operated upon a second time, when a ruptured extrauterine pregnancy was found. In an abdominal pregnancy, which was operated upon, the only criticism that can be made is that no preparation was made for a transfusion. In the final case, in which there was no delay in sending the patient to the hospital or in operating, the operator thought he was taking out the left appendages, but examination of the specimen showed that he had done a hysterectomy. The left horn of a bicornuate uterus had ruptured, and in spite of two transfusions this woman promptly died.

The delay in sending the patients to the hospital was unquestionably responsible for 3 of these deaths, but whether it was due to the physicians' delay or to the patients' delay is undetermined. The other cases we do not feel could be regarded as preventable.

There were no cases assigned to 143, "Other accidents of pregnancy".

Under rubric 144a, "Puerperal hemorrhage, placenta praevia," there were 9 cases (fig. 2).

144a. PLACENTA PRAEVIA

	No.	Man. dil. and del.	Cesarean section	Braxton-Hicks	Undel'd
1933	6	3	2	0	1
1934	2	0	1	1	0
1935	1	0	1	0	0
Total	9	3	4	1	1

FIGURE 2.

We call your attention to the fact that this does not mean that there were only 9 placenta praevias in this list of deaths, but that only 9 could be rightfully assigned to placenta praevia as the chief cause of death. In 3 cases there was manual dilatation and delivery as soon as the doctor saw the patient. Four had cesarean sections. There was 1 Braxton-Hicks version, and 1 died undelivered. In 2 the cesarean sections were promptly done, but uncontrollable bleeding occurred and in spite of transfusion they died. In 1 of these a hysterectomy was necessary. In the third cesarean section, a patient who had had a previous section, the intestine was accidentally opened. The patient died apparently from some obscure cause; it was not sepsis. In another case the family physician had kept the patient in bed for two weeks, during which time she had had repeated vaginal hemorrhages. She was found by the ambulance surgeon in a pool of blood. She was taken at once to the hospital, and an immediate cesarean section was done, but in spite of two transfusions she died. The 1 case that died undelivered began bleeding at seven months. She was put

to bed, was not examined, and in the eighth month she had one tremendous hemorrhage and died shortly after reaching a hospital.

In regard to the preventability of these deaths, delay in operating was a major factor in 2 cases. Whether the manual dilatation and delivery was the cause of 3 of these deaths it is difficult to say, but we feel that such treatment is not the proper method of managing placenta praevia. The remaining cases apparently were unpreventable.

To 144b, "Other puerperal hemorrhages", are assigned 12 cases (fig. 3). Four were normal deliveries, 3 were forceps, 4 cesareans, and 1 had a Voorhees' bag inserted, followed by a version. Nine of these died from uncontrollable postpartum hemorrhage. Two had accidental hemorrhages, and in 1 the type of bleeding was not definitely determined. Again, we feel it is difficult to say whether these cases were preventable, but it is a fact that 6 of the postpartum hemorrhage cases had no transfusion. We appreciate that at times the hemorrhage may be so sudden that a transfusion is impossible, but we do feel that in accidental hemorrhages where time is taken to prepare for a cesarean, there is also time to obtain donors for a possible transfusion. In 2 of these cases no attempt was made to transfuse the patients, and these must be definitely regarded as preventable deaths and evidence of poor obstetrics.

144b. OTHER PUERPERAL HEMORRHAGES

	No.	Normal	Forceps	Cesarean section	Bag and version	Postpartum	Accidental hemorrhage	?
1933	3	1	1	1	0	2	0	1
1934	5	2	2	1	0	5	0	0
1935	4	1	0	2	1	2	2	0
Total	12	4	3	4	1	9	2	1

FIGURE 3.

Under 145a, "Puerperal septicemia", there were 74 deaths, 26 per cent (fig. 4). There were 20 normal deliveries, 22 operative deliveries (forceps, breech or version), 30 cesarean sections, 1 vaginal cesarean, and in 1 case the method of delivery was unrecorded. It is difficult to understand why so many normal deliveries died. There is a possibility of a hospital epidemic accounting for 3 of the deaths. A history of sore throat and coryza may be a possible cause of 2, but for the remainder of these normal cases there is no obvious cause. Lack of proper technic must be assumed. A gas bacillus infection occurred in 1, following a breech extraction. In 1 case 0.5 cc. of pituitrin was given with the os not dilated, a normal delivery followed and a severe postpartum hemorrhage took place, which necessitated a hysterectomy. In another, 2 minims of pituitrin were given a patient by her doctor at home, she immediately went into hard labor and complained of severe pain. She was etherized so that she could be taken to the hospital. When she arrived there, a transverse presentation was found, and a version immediately done. Examination after delivery showed a rent in the left uterine segment. The placenta was left *in situ*. She was immediately operated upon, and a rupture of the uterus into the broad ligament was found. A hysterectomy was done at once, but she died six days later of general peritonitis. In another case a vaginal section was done on a patient four months pregnant for a therapeutic abortion. During the operation a loop of intestine was brought down; it was finally recognized, and a consultant was called in. The patient was operated upon at once, and an intestinal resection done. In another case the woman gave a history of amenorrhea of five months' duration. The uterus was about the size of a three months' pregnancy. A hysterectomy and appendectomy were done under spinal anesthesia. Twenty-four hours later she showed signs of sepsis and promptly died.

These last cases must be regarded as preventable. The use of pituitrin in a case with an undilated cervix, the perforation of the uterus, the bringing down of a loop of intestine and the performance of a hysterectomy for a blighted ovum are all evidences of very bad obstetrics. That 20 operative delivery cases should have died allows of but one conclusion—the operators' technic was poor.

145a. PUERPERAL SEPTICEMIA

			Forceps					Cesarean				
	No.	Normal	Low	Med.	High	Breech	Version	Classical	Low	Vaginal	Hysterectomy	No record
1933	24	10	2	1	0	1	1	5	3	0	1	1
1934	28	6	5	2	1	0	2	7	5	1	1	0
1935	22	4	3	0	1	0	3	4	6	0	2	0
Total	74	20	10	3	2	1	6	16	14	1	4	1

FIGURE 4.

The cesarean sections that are classified as septic we prefer to take up later in the analysis.

The next rubric is 146, "Puerperal albuminuria and eclampsia". Under this there were 32 deaths. The prenatal care was good in only 7 of these cases. In the others there was none at all or it was wretched. Nine of these patients died undelivered. One died of postpar-

tum eclampsia. Thirteen patients were sent to the hospital in coma or had had convulsions before entrance to the hospital. One of these had had a convulsion two weeks before she was sent to the hospital with a high blood pressure and symptoms of impending eclampsia. Nine had normal deliveries, 1 of these being the postpartum eclamptic case. Four were delivered by forceps. One was an accouchement forcé, and in 6 a cesarean section was done. In 1 the method of delivery was unrecorded. All but 7 of these cases were abominably neglected, but for the greater part of the neglect the patients themselves were to blame. They sought no medical care until desperately ill. Eclampsia occurred in 1 of the 7 cases that had good prenatal care, but in spite of treatment the patient died. Included in this group because of the whims of the International Classification were 2 cases of pyelitis. The treatment that the undelivered women received in the hospitals depended entirely upon what hospital they happened to enter. Some were treated by the Stroganoff method and others by plasmapheresis, colonic irrigation and sedatives.

Until it becomes the established custom that all pregnant women are under competent medical supervision we shall continue to have deaths from eclampsia. Had these patients had such supervision, the number of deaths in this rubric would have been greatly diminished.

Under rubric 147, "Other toxemias of pregnancy," there were 19 deaths. Eight were typical cases of nausea and vomiting of pregnancy, 4, in all probability, had chronic nephritis, 5 had toxemia of pregnancy, in 1 the death certificate was signed "acute yellow atrophy" and 1 had long-standing hypertension. Nine of these cases died undelivered. Six of these occurred in hospitals where therapeutic abortions are not allowed.

There was 1 neglected hypertensive case, and in 2 cases the women were found dead. The autopsy in 1 of these showed a condition consistent with a toxemia of pregnancy, while the second showed marked waxy edema. The death certificate of the latter was signed by the Boston Board of Health, in all probability a long-standing nephritis. In 2 cases there was definite delay in emptying the uterus while in the hospital, and in 2 others the patients refused for some weeks to enter a hospital for treatment.

An excellent example of the results of serious delay that may occur in a hospital where there is no obstetric service is the following case. The patient, 25 years of age, had had three children. She had been vomiting for six weeks and had lost thirty pounds in weight. She had been in bed for two weeks under the charge of her own physician, when she was sent into a hospital for care. She left this hospital and stayed at home a short while, then was sent into a hospital that had no obstetric service. She had menstruated only twelve times in her life. The diagnosis was probable ovarian dysfunction. Four days after entrance the Aschheim-Zondek test was positive. There is no report in the record of a satisfactory vaginal examination. She complained of blurred vision. Her eyegrounds were very carefully studied, gastrointestinal x-rays were taken, and ten days after all possible tests were done, a consultation with an obstetrician was requested. He immediately advised a therapeutic abortion because of pernicious vomiting. She was transferred to the surgical service and curetted. She went into very serious shock following operation, was transfused and gradually became worse, dying two weeks later. This seems to have been a preventable death.

Another typical example in this group is the following case. The patient, a primigravida, had a mild toxemia of pregnancy with a systolic blood pressure of 146 mm. and a very slight trace of albumin. It was thought necessary to induce labor. She was first given castor oil and quinine, without effect. The membranes were then ruptured. Labor did not start, and finally a Voorhees' bag was inserted. When this was expelled, a difficult mid-forceps was done. The patient went into profound shock after delivery, the baby lived only a few hours, and the mother steadily failed in spite of transfusion and died. Whether this woman should have been induced for what was apparently a mild toxemia is open to doubt. The results obtained surely did not warrant it.

We shall continue to have deaths in this group until we can improve our treatment of the toxemias of pregnancy and while some of the hospitals forbid therapeutic abortions.

The next group, rubric 148, "Puerperal phlegmasia alba dolens, embolus, sudden death," contains a surprisingly large number of deaths, 44. Of these, 30 followed some form of operation. Nine followed normal delivery, 1 assigned by certificate only. Four were undelivered. In 2 of the undelivered cases it is only known that the patients died suddenly. The death certificates were signed by an agent of the Department of Health. Another had an antepartum embolus, and the final undelivered patient died during the second stage of labor from a probable rupture of a large vessel. One patient died from an air embolism during the intravenous administration of glucose, and 4 died from anaphylactic shock. Four of these patients had been operated upon. Three had had cesarean sections, 1 a dilatation and curettage, and 1 had had a normal delivery. Also included in this group is 1 case that died of an embolus following, as the physician testified, an attempt to replace a ret-

roverted nonpregnant uterus with an instrument. The physician was tried for malpractice. The patient was some three months pregnant and during his manipulation died suddenly on the table in his office. He was acquitted of the charges brought against him. One patient died and we could not find out whether she was operated on.

It is impossible to say how many of these cases should be regarded as preventable. A few were septic and were allowed to get up too soon, but in the great majority there was nothing to suggest that an embolus might occur. The air embolus should not have occurred. We appreciate that even when the matching of blood is carefully done, accidents do occur, but it is surprising that 4 cases should have died of anaphylactic shock.

A whole evening could profitably be spent on a careful, minute analysis of the deaths that followed cesarean section, but we shall report only the important facts. We have placed under rubric 149a only 11 cases as deaths caused by cesarean section. We placed these 11 in this rubric because no other place seemed to be satisfactory. In 1 case the doctor had disappeared, and in a second the doctor had died before the case was investigated. Four had sections done because it was considered the safest method of delivery in severe cardiac cases. In 1 there was an intestinal obstruction due, presumably, to a previous surgical procedure, but, if this cesarean section had not been done, death might not have followed. However, because of a contracted pelvis it had to be done, and there was no reason to expect that this complication would occur. In another, a repeat section was done, and the next morning the patient had a fulminating lobar pneumonia and died. One case developed a general peritonitis immediately following operation, and on autopsy it was found that she had a ruptured appendix abscess. In another case there was a definite upper respiratory infection, spinal anesthesia was attempted but failed, and she was given gas-oxygen and ether for the operation. The final case, which has been assigned to this rubric, is one that had had two attempts at operative delivery outside the hospital. She had had four previous normal deliveries. One doctor replaced a prolapsed arm and cord, but was unable to deliver the patient, and another doctor was called, who attempted to apply forceps. This was unsuccessful. The patient was then sent into a hospital, a low cervical section was done at once, and a stillborn baby was delivered.

If we could stop here, without further report on the cesarean sections, it would be well. But that is impossible. We found that, in all cases, 78 sections had been done, 47 of the classical type, 28 of the low, and in 3 the type was unrecorded (fig. 5). Fifteen of these were done as repeat sections, 15 were done for disproportion, 11 for bleeding, 12 for toxemia or eclampsia, 4 were done on elderly primigravidae, 1 on a primiparous breech, 1 by election of the patient, 2 each for contraction ring and pyelitis, 10 for cardiac conditions, 1 for poor labor, 2 for a rigid cervix, 1 because there had been a previous obstetric disaster, and in the final case a section was done in the hope of obtaining a live baby, although premature, in a patient that had a brain abscess.

Whether all these indications were true and sufficient it is difficult to determine. Disproportion as an indication depends entirely upon the physician's judgment. It may have been present, but, when it is known that a certain number of these cases were done by men who have had absolutely no training in obstetrics, the indication is open to question. Several of the eclamptic cases were operated on without any previous treatment. This cannot be said to be good obstetrics. It is impossible to say whether the bleeding cases were best managed by cesarean section. We do, however, believe that a greater effort should have been made to transfuse some of these patients. Election by the patient can scarcely be regarded as an adequate indication!

CESAREAN SECTION

TYPE AND INDICATIONS

	Total	Classical	Low	Unrecorded	Disproportion	Repeat	Bleeding	Eclampsia	Toxemia	Elect. of patient	Elderly primip.	Breech	Brain abscess	Contraction ring	Pyelitis	Cardiac	Poor labor	Rigid cervix	Previous disaster
1933	27	19	8	0	5	6	4	1	4	1	1	0	1	2	2	0	0	0	0
1934	28	16	9	3	5	5	4	0	2	0	2	1	0	0	0	7	1	1	0
1935	23	12	11	0	5	4	3	4	1	0	1	0	0	0	0	3	0	1	1
Total	78	47	28	3	15	15	11	5	7	1	4	1	1	2	2	10	1	2	1

FIGURE 5.

The chief cause of death in these cases was sepsis, which accounted for 31 deaths, or 40 per cent (fig. 6). Embolus came next with 13 deaths. The others were due to the various causes connected with the indications for the procedure. The case assigned to scarlet fever as the cause of death occurred in an elective case. The rash appeared twelve hours after operation.

We wish to call to your attention the following cases. In 1, the woman, six and a half months pregnant, went into labor. No dilatation of the cervix occurred. After forty-eight hours of labor it was discovered that she had had a high amputation of the cervix some years before. A classical cesarean section was done at once. Had the physician in charge obtained a proper history from the patient, this fatality could have been avoided.

Three patients had sections following attempted operative deliveries before entering the hospital. We have already described 1. On the second an attempt at delivery was made at home for several hours before she was sent into the hospital. The hospital record says: "Marked ecchymosis of the anterior vaginal wall, a moderate amount of labial edema; on vaginal examination a caput was found, but the biparietal diameter was not through the ischial spines." A low section was immediately done. In the third case an attempt was made to deliver from below by forceps, but the second blade could not be applied, and the patient was taken into the hospital and a cesarean section performed. Two of these 3 patients had stillborn babies. The only excuse for doing a section in these cases was the hope of obtaining a live baby and one that would survive. It seems that too great emphasis was placed on the survival of the baby.

A physician that does a cesarean section because of the patient's desire and then loses the patient from general peritonitis lays himself open to very serious criticism. We ask you to take note that of the women that died in this investigation 27 per cent, or 1 in 4, died after having had a cesarean section. We do not believe that there is any justification for so many sections.

Rubric 149b, "Other accidents of childbirth", contains 31 deaths (fig. 7). In this list there were 6 cases of ruptured uterus. One occurred in the scar of a previous cesarean section. The others followed hard, operative deliveries. Two cases are typical examples of this group. In the first, three unsuccessful attempts at delivery had been made before the patient was sent to the hospital. She entered the hospital with the head in sight. She had a very narrow outlet, and an attempt was made in the hospital to deliver her by forceps. This was unsuccessful, and a version was attempted. She was finally delivered, but died before the completion of the third stage. A second case was sent into the hospital by a private physician with a diagnosis of transverse position. The patient in the hospital admitted that she had attempted to replace a prolapsed arm and cord hours before the physician arrived. He then had attempted to deliver her but was unsuccessful and sent her into the hospital. When she arrived in the hospital, forceps delivery was attempted but failed, and then a version was done. A manual extraction of the placenta revealed a rupture in the left uterine segment.

CESAREAN SECTION—CAUSE OF DEATH

	Total	Sepsis	Embolism	Anaphylaxis	Placenta praevia	Sep. placenta	Pyelitis	Toxemia	Eclampsia	Respiratory inf.	Rupt. appendix	Intestinal obstr.	Brain abscess	Scarlet fever	Postpartum hem.	Cardiac	Cesarean section	Undetermined
1933	27	9	4	0	2	1	2	1	2	1	0	1	1	1	0	0	1	1
1934	28	12	3	2	1	0	0	1	0	0	1	0	0	0	1	4	0	3
1935	23	10	6	0	1	2	0	0	3	1	0	0	0	0	0	0	0	0
Total	78	31	13	2	4	3	2	2	5	2	1	1	1	1	1	4	1	4

FIGURE 6.

149b. OTHER ACCIDENTS OF CHILDBIRTH

	No.	Rupt. uterus	Shock	Lung abscess	Anesthesia	Inverted uterus	Cardiac failure	Surg. op.	Resp. infec.	Certif. only
1933	11	4	3	2	1	1	0	0	0	0
1934	10	2	1	0	3	0	1	1	1	1
1935	10	0	2	1	4	0	1	1	1	0
Total	31	6	6	3	8	1	2	2	2	1

FIGURE 7.

She was operated upon at once but died twenty-four hours later.

There were 6 cases assigned to shock as the cause of death. In these cases it is definitely recorded that there was no ruptured uterus. There were 13 cases in which anesthesia apparently was the cause of death. Three developed lung abscesses, and in 4 the death was the direct result of the inhalation of vomitus. Three vomited a great deal during the anesthesia and died of an inhalation pneumonia.

NONOBSTETRIC DEATHS

	Total	Pneumonia	Appendicitis	Cardiac	Brain abscess	Erysipelas	Scarlet fever	Tuberculosis	Cholecystitis	Carcinoma	Acute alcoholism	Ruptured spleen
1933	22	14	2	1	1	0	1	1	1	0	1	0
1934	6	2	0	1	0	0	0	1	0	1	0	1
1935	6	5	0	0	0	1	0	0	0	0	0	0
Total	34	21	2	2	1	1	1	2	1	1	1	1

FIGURE 8.

Three died following anesthesia given with an upper respiratory infection. There was 1 inversion of the uterus, and 2 died from apparent cardiac failure during delivery. There were 2 deaths following a surgical procedure, 1 of which was for removal of a dermoid cyst. The patient shortly after operation started in labor and delivered herself. There may have been a low-grade sepsis, but it is not certain. The second patient had a normal delivery and twenty-four hours later developed abdominal pain with repeated vomiting and abdominal distention. An intestinal obstruction was diagnosed, and a cecostomy was done. There was some mucopurulent free fluid in the abdomen. She died eight days after operation. The final case is put into this group by the certificate only.

We feel that it is difficult to say exactly how many of these cases were preventable, but there is no question that careless anesthesia was responsible for at least 10 of these deaths. The cases of ruptured uterus were unquestionably evidence of poor obstetrics. It is impossible to say whether the other cases were preventable.

The final rubric, 150, "Other and unspecified conditions of the puerperal state", contains 1 case. This patient was discharged from the hospital well on the fourteenth day, and six weeks after delivery she developed a mastitis following a marked pharyngitis. She had an overwhelming septicemia and died on the eighth day.

We now come to a group of deaths in which pregnancy was present but could not be regarded as the chief cause of death (fig. 8). There were 34 cases in this group. Twenty-one of them had lobar pneumonia or bronchopneumonia with a pregnancy of from two months to full term. Some died undelivered and others miscarried just before death, but it was clear in all of these that pneumonia was the main factor in the death. There were 2 cases that were operated on for acute appendicitis in early pregnancy. There were 2 cases that died apparently as a direct result of their cardiac condition early in pregnancy. There was 1 brain abscess case in which a cesarean section was done shortly before death. One woman died of erysipelas, and 1 developed scarlet fever twelve hours after delivery. There were 2 cases of advanced tuberculosis, 1 of gallbladder disease, which was operated on, 1 of carcinoma of the cervix, 1 woman was found dead in bed from acute alcoholism with the baby partially delivered and the final case was one of rupture of the spleen from unknown cause. This patient was operated on, but the hemorrhage was uncontrolled and she died. None of these deaths can be definitely attributed to the puerperal state.

We have mentioned under the various rubrics that cases died undelivered. In all there were 33 such cases, a large number for such a series. The explanations for them we have already given.

The next chart (fig. 9) shows the number of births and deaths as given to us by the Boston Department of Health. In order that our study would be as complete as possible, we went through the death certificates filed at the State

TOTAL BIRTHS, STILLBIRTHS AND MATERNAL DEATHS WITH MORTALITY RATES

			Deaths		Per 1,000	
	Births	Still-births	Official	Study	Official	Study
1933	16,239	488	104	127	6.2	7.5
1934	15,818	445	87	97	5.3	5.9
1935	15,835	403	87	94	5.3	5.7
Total	47,892	1,336	278	318	5.6	6.4

FIGURE 9.

House, and it is from these records that we found our additional cases. This explains our higher death rate, for we have included all deaths of patients that were pregnant which occurred in the years studied. Had we eliminated the 40 deaths, the death rate would have been, according to our figures, 5.6.

The changes in classification from those of the official figures (fig. 10) were due largely to the fact that we had better understanding

DISEASES OF PREGNANCY, CHILDBIRTH AND THE PUERPERAL STATE

Rubric	1933		1934		1935	
	City	Study	City	Study	City	Study
140 Abortion with septic conditions	8	9	11	11	13	16
141 Abortion without mention of septic conditions (to include hemorrhage)	3	2	0	0	1	1
142a Ectopic gestation with septic conditions specified	1	3	0	1	1	1
142b Ectopic gestation without mention of septic conditions	7	5	0	0	2	2
143 Other accidents of pregnancy (not to include hemorrhages)	1	0	2	0	0	0
144a Puerperal hemorrhage—Placenta praevia	8	6	3	2	5	1
144b Puerperal hemorrhage—Other puerperal hemorrhages	6	3	6	5	4	4
145a Puerperal septicemia (not specified as due to abortion)—Puerperal septicemia and pyemia	21	24	26	28	23	22
146 Puerperal albuminuria and eclampsia	13	16	7	6	15	10
147 Other toxemias of pregnancy	10	7	6	7	1	5
148 Puerperal phlegmasia alba dolens, embolus, sudden death (not specified as septic)	7	14	5	15	4	15
149a Other accidents of childbirth—Cesarean operation	9	4	13	6	4	1
149b Other accidents of childbirth—Others under this title	10	11	8	10	13	10
150 Other and unspecified conditions of the puerperal state	0	1	0	0	1	0
Additional nonobstetric deaths included in study	—	22	—	6	—	6
Totals	104	127	87	97	87	94

FIGURE 10.

of the cases, because of our interviews, than was revealed by the death certificates. One classification used in the official figures was 194b. This refers to violent and accidental deaths. It does not seem reasonable to use this rubric in describing deaths occurring during pregnancy or the puerperal state. Also, asphyxia by vomiting is assigned to 194a. Both these classifications lose sight of the pregnant or puerperal state, and unless each death certificate is carefully studied the cases would not be included in any study of maternal deaths.

We are unable to go into the intricacies of the International List of Causes of Death. There should, however, be some simple way to show accurately the true picture of chronic nephritis and cardiac disease in relation to pregnancy. We all appreciate the fact that, no matter how good the medical care may be, deaths do occur because of these complications. The inclusion of pyelitis with puerperal albuminuria and eclampsia is totally unwarranted and should be changed at some future revision.

We feel that there should be few or no deaths classified under 149a, "Cesarean operation." Cesarean section alone should not be accepted on a death certificate as the chief cause of death, except in rare instances. The underlying cause should be given. In many cases in our study this was omitted and was found only on searching the hospital records and on interviews with the physicians.

As long as women suffering from nephritis or severe cardiac or other organic diseases become pregnant there will be deaths occurring in this group. Even when therapeutic abortions are done, some deaths may occur. The management of these cases brings up debatable points, which we do not care to enter upon. The medical profession should not, however, be blamed for these deaths.

It appears that, in spite of attempts to educate the laity to the need of prenatal care, many of these women received none at all, and in some cases where care was sought it was inefficient. As discouraging as this condition is, the constant reiteration of this need must be continued.

The lack of preparation for transfusions was evident, and the deaths from anesthesia poorly administered were too frequent. These are two possible preventable causes of death that should be speedily corrected.

It is difficult to know what to say in regard to sepsis. The respiratory infections unquestionably had something to do with these deaths. The technic that was employed in the deliveries is difficult to evaluate. It cannot have been so good as it should have been.

That any such number of cases with cesarean

operations should have died is evidence of bad obstetrics. Elective cesarean sections were done for specious reasons. Some were done on badly infected cases. It is interesting to note that no craniotomies were found in this whole series.

A report such as this is depressing. Good obstetrics is practiced in Boston, but this report brings out the fact that there is also a good deal of bad obstetrics. The results of this study are a direct challenge to those men that have done poor work, and it is their duty to perform, in the future, only work that they are capable of doing. The report is a direct warning to all physicians that improperly selected operations or operations badly performed carry with them a high maternal mortality. It is also a direct challenge to the hospitals that have allowed bad obstetrics to be practiced by members of their staffs.

NIGHT BLINDNESS DUE TO VITAMIN A DEFICIENCY: A CONSIDERATION OF ITS IMPORTANCE IN TRAFFIC PROBLEMS*

BY HAROLD JEGHERS, M.D.†

NIGHT blindness, often called hemeralopia, dysaptatio visualis, or erroneously, nyctalopia, is the difficulty, and occasional inability, of adapting the faculty of vision to very faint illumination. While this condition may result from various intraocular diseases, such as glaucoma, detached retina, choroiditis, retinitis pigmentosa and optic neuritis, it may also occur without any ophthalmoscopically demonstrable lesion being present in the eye and is then termed *essential* or *idiopathic hemeralopia*.[1, 2] To establish satisfactorily the presence or absence of this type of hemeralopia‡ requires the performance of a test not commonly used at present.

In a recent paper on the subject, Frandsen[1] summarizes the objective data concerning essential hemeralopia as follows: "These patients show an increased minimum light visible. In *reduced illumination* the acuity of vision is lowered, the field of vision is narrowed for white as well as for colored objects, so that the field of vision for blue in higher degrees of hemeralopia becomes smaller than the field for red. Dark scotoma appears at higher clarity than in the case of normal light sense, the adaptation time is prolonged, the power of distinction is lowered—all signs which signify that the range of adaptation is reduced."

These objective manifestations can be quantitatively measured and constitute the means available at present of detecting mild to moderate degrees of night blindness. The practical aspects of these different methods are discussed elsewhere.[3]

There is now abundant evidence available to show that in older children and adults essential hemeralopia is usually the earliest and the most constant manifestation of avitaminosis A[4, 5] and that it may exist for years without any other sign of the deficiency appearing.[5, 6] Only severe degrees of night blindness annoy the patient sufficiently to cause him to seek medical attention.[7] Such cases, however, are fairly uncommon as compared with the milder to moderate forms of hemeralopia.[1] It is only beginning to be appreciated that a mild to moderate degree of night blindness may be present and interfere with the efficacy of dark adaptation without the patient being aware of its existence.

Vision under faint illumination depends upon the metabolism of the visual purple of the retinal rod cells. The visual purple becomes exhausted when the eye is exposed to bright light (sunlight or artificial light) and rapidly regenerated if an adequate supply of vitamin A is present in the body. Vitamin A has been shown to be the precursor of visual purple[8, 9] and it is present in very large amounts in the retina.[10] A deficient intake of this vitamin causes the body stores to be low, and hence is the cause of poor regeneration of visual purple. An inadequate supply of regenerated visual purple in the retina, in turn, causes poor vision, hemeralopia, under faint illumination.[8, 9]

For many years foreign investigators have been using various types of photometers to measure quantitatively minor to moderate degrees of night blindness.[11] It was also discovered empirically that hemeralopia not due to intraocular disease would respond to food rich in vitamin A, with a subsidence of the symptoms and a return of the photometer reading to normal.[11, 12]

The experimental demonstration by Fridericia and Holm[8] and Tansley[9] that vitamin A was necessary for regeneration of visual purple, along with the numerous clinical studies showing the relation between gross hemeralopia, xerosis conjunctivae and keratomalacia, and avitaminosis A,[13, 14] led to the logical conclusion

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‡Hemeralopia and night blindness, used interchangeably for the remainder of this article, refer to the essential or idiopathic hemeralopia due to vitamin A deficiency.

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that such photometer tests could be used to detect subclinical degrees of vitamin A deficiency. This was amply shown to be so by Frandsen[1] and others in Europe and Jeans and Zentmire[15] in this country.

In their original study, Jeans and Zentmire[15] found 21 per cent of a large group of children to have slight to moderate degrees of vitamin A deficiency. In other more extensive surveys among children, these same investigators[16] obtained figures that varied from 26 to 79 per cent, depending on the economic level of the group studied and whether urban or rural communities were used. Sandler[17] likewise found many children that were deficient.

Park[7] found essentially the same to hold true for adults. About 700 persons were tested. Jeghers,[3] in studying only adults, found mild to moderate degrees of vitamin A deficiency present in from 10 to over 50 per cent of each group tested. Variation in percentage of deficient persons in each group depended upon several factors, which are discussed elsewhere.[3]

In practically every instance, where tried, each of these investigators was able to bring the photometer reading to normal by oral vitamin A or carotene medication for two weeks or longer, at the same time causing the hemeralopia that had been present to disappear entirely. These investigations, and the even more extensive ones in Europe,[1] show that a significant number of supposedly healthy persons have a mild to moderate, and occasionally severe, degree of vitamin A deficiency. An important conclusion is that this large group, without in most cases being aware of it, has vision that, while normal daytimes, is inefficient in dim illumination. The degree of this inefficiency varies, of course, directly with the degree of vitamin A deficiency that exists.

A point of great practical and economic importance arose while the writer was testing supposedly normal adults for avitaminosis A, using a Birch-Hirschfeld photometer.* Questions were asked of each person that gave a low photometer reading in order to determine if their diminished dark adaptation caused them any trouble. Quite striking were the stories that several vitamin A deficient persons told relating to the difficulty they experienced when driving an automobile at night. This difficulty was much more noticeable when driving in the country where there were no street lights. Passing an oncoming automobile was particularly difficult because of the temporary blindness caused by the glare of headlights. The following case histories are significant in this regard.

CASE 1:

A male laborer, 34 years old, whose photometer reading was 40 per cent below normal, mentioned that driving an automobile at night was a definite strain as compared with day driving, and the situation seemed to be gradually getting worse. It took much attention on his part to be sure where the edge of the road was, and a person walking in the dark was dangerously near the automobile before being noticed. The lights from passing cars dazzled him very easily. No accidents had occurred, but were feared. Questioning revealed an obviously deficient diet. There was no organic disease present to cause vitamin A deficiency. His eyes were normal and vision in daylight was good. There was no evidence of xerosis of the conjunctivae or changes in the skin. Carotene* medication, 20,000 U. S. P. units daily for three weeks, along with a change in diet caused the photometer reading to return to normal. Very definite improvement in his ability for night driving was noticed. He was advised to take vitamin A concentrates regularly.

CASE 2:

This patient, a 31 year old, married, certified public accountant, is reported through the courtesy of Dr. Henry Bakst who, suspecting vitamin A deficiency, requested a photometer test.

About five years ago, fearing a gain in weight, the patient eliminated all dairy products and many vegetables from his diet. He lived continuously on this deficient intake of vitamin A. Shortly after starting this regime, his skin gradually became dry and rough and, finally, began to itch. His hair became dry, brittle and lusterless and fell out in increasing amounts. Hordeolums appeared from time to time. On entering the darkened interior of a motion picture theatre the patient experienced great difficulty in reaching his seat and had to be guided by his wife. The pictures seemed blurred for five or ten minutes and then cleared. Driving an automobile at night became difficult, especially when trying to pass an oncoming car. Even dim headlights dazzled him. Under such circumstances, the patient drove, as he put it, "entirely by instinct". He feared an accident. One night, a police officer stopped him and threatened arrest for "drunken driving", which amazed the patient since he had not been drinking. He had been driving on the wrong side of the road, and was entirely unaware of it. When the streets or roads were illuminated by street lights, driving was much easier than on dark, unlighted country roads. Several eye examinations had revealed 20/20 vision. Many forms of therapy had been tried for his skin lesions without relief. His general health was fair.

Examination showed his hair to be sparse and brittle and the scalp dry. The palpebral conjunctivae were dry, while a slight degree of xerosis was present on the bulbar conjunctivae. His skin was dry over the entire body, excoriated from scratching and revealed marked hyperkeratosis follicularis. There was no evidence of systemic or intraocular disease. Routine laboratory studies were normal. When examined in the dark room the patient was unable to see a luminous watch dial after ten minutes (normally seen in three minutes) and gave a photometer reading 60 per cent below normal.

Carotene (30,000 units daily) was prescribed, and butter, milk and raw carrots were added to his diet. Striking changes followed. Itching ceased within a few days. Over a period of weeks his skin became soft and smooth, the xerosis disappeared, his hair ceased falling out and the scalp became oily. At the end of three weeks the patient was again tested in the dark room. He could now see

*The Birch-Hirschfeld photometer used in these studies was of Zeiss manufacture.

*Carotene used in these studies was generously supplied by the S. M. A. Corporation of Cleveland, Ohio.

the luminous watch dial in four minutes, and his photometer reading was only 10 per cent below normal. Improvement remained permanent. With the return of the photometer reading to normal, his former difficulty in adapting his vision to dim illumination disappeared.

Two other persons were found that had a complaint similar to case 1, but unfortunately they were not available for a follow-up study with the photometer. There were, in addition, at least a dozen persons with low photometer readings, who were drivers of automobiles. None of these persons, however, were aware that they had a mild to moderate degree of hemeralopia.

This fact was not appreciated and searched for until near the end of the series of tests, otherwise the number would undoubtedly have been greater. It so happened that most of the subjects tested were nurses and students who did not own or drive an automobile.

Being impressed with the possibility that mild to moderate degrees of hemeralopia due to vitamin A deficiency could be responsible for some of the automobile accidents that occur at night, a search was made for more evidence on the subject. A review of the available medical literature and a theoretical consideration of the subject under question offer much to support this contention.

Since suitable facilities to study this problem were not available, actual field studies to prove this point were not attempted. A review of the subject is being presented with a twofold purpose in mind: (1) to point out that there is already enough evidence available to make the subject far from a theoretical one and (2) to try to stimulate interest in the general medical profession, as well as traffic commissions and automobile associations throughout the country.

The subject is not a new one and has been mentioned several times in the literature, but evidently the practical possibilities of this problem have been overlooked.

Vignalou,[18] in 1933, reported the case of a man, who had had repeated automobile accidents at night and who was found to have night blindness. It was pointed out that the French laws completely overlooked hemeralopia as a cause for refusing a driver's license. A plea was made that the government consider the problem. This investigator called attention to the need of using a photometer or the performance of visual fields in dim illumination to detect this condition.

Rollet,[19] a year later, wrote of two chauffeurs, whom he had examined and found to have essential hemeralopia. Because of the normal eyegrounds they were only detected by means of a radioluminescent photometer. These two men had had a total of six accidents at night with loss of human life, whereas they were noted for their skill in driving during the daylight. The medicolegal aspect of this subject was again discussed. Neither of these authors mentioned the relation between vitamin A deficiency and hemeralopia.

A most interesting case of nutritional night blindness in this country was diagnosed by Wilbur and Eusterman.[20] The patient, a male of 48 years, had persistent diarrhea due to a gastrocolic fistula, but was, however, ambulatory. In spite of an adequate diet, the frequent bowel movements led to vitamin A deficiency and night blindness. One of his major complaints was difficulty in driving an automobile at night. He was often unable to tell whether he was on the shoulder or the wrong side of the road and he had had numerous mishaps. After repair of the fistula and the use of a generous diet, all his symptoms of night blindness disappeared. Examination of the eyes was normal except for mild sclerosis of the retinal arteries. Perimetric fields were reported as normal, but were probably done in good illumination. This, of course, represents a very severe case and the night blindness probably existed for many months before it became serious enough to make the patient aware that something was wrong.

Tilderquist[21] had a patient, a young male, aged 21 years, who complained that he could not see well after dark. Difficulty in driving an automobile at night because of inability to see the road was the first and most prominent manifestation of night blindness in this instance. Because of fear of an accident, driving was discontinued. The physical examination was normal. There was 20/20 vision in each eye and the fundi were normal. The only objective finding was a reduction in the visual fields. The diet of this patient was found to be deficient in foods rich in vitamin A. Following a change in his eating habits, the night blindness disappeared promptly.

Park[7] has a much more significant communication. In a study of 275 persons with a Birch-Hirschfeld photometer he found that only twelve of them complained of night blindness, whereas more than half had hemeralopia as shown by this instrument. Of special interest here is the story of two of these patients, who were automobile drivers and had a severe enough degree of night blindness to be aware of it themselves. One patient was a male, aged 42 years, in good health, who complained of difficulty in driving at night and that the lights from other cars annoyed him greatly. A very low photometer reading was secured. It was discovered that his diet was inadequate. Complete relief of his night difficulties followed a good diet and carotene medication for a few months. The photometer reading returned to normal. The second person was a female, aged 35 years, also in good health, who for six months had noticed a progressive difficulty in driving her automobile at

night. Because of the constant fear of collision she gave up driving altogether. The initial photometer reading was less than 50 per cent of normal. After carotene therapy for several weeks the photometric reading returned to normal, and she was again able to drive with ease at night. Park mentions the significance of a hemeralope driving at night, and again stresses that such a person is in more danger of having an accident than a person with normal dark vision.

Frandsen's[1] careful case records of sixty-six hemeralopes studied in Denmark, where bicycling is the common method of transportation, add a bit more support to the subject under discussion. A common complaint of these patients was marked difficulty in riding a bicycle at night. After medication with vitamin A, this difficulty disappeared in each case.

It has been shown, in experimental animals[8] as well as in human beings,[5] that hemeralopia becomes manifest in vitamin A deficiency only after the eye has been exposed to light. A person who remains in a dimly lighted room all day needs but little visual purple to carry on his visual needs. Exposure to daylight and artificial light is, of course, what uses up the store of visual purple. Excessive exposure to light will use it up more rapidly.

This fact was well demonstrated by Aykroyd,[5] who found that it was the custom among some persons in Labrador, where severe vitamin A deficiency was common, to wear a bandage over one eye, while the other eye was left uncovered, in order to see to carry on the day's activities. After dark they would remove the bandage. The eye that had been exposed to the sunlight was night-blind, but the other eye, which had been covered, was not affected and served them until the next morning, when the process was again repeated.

In night driving we have what amounts to a perfect experiment to bring out hemeralopic manifestations, if vitamin A deficiency exists. The glare from the lights of each oncoming car depletes a fraction of the visual purple. If the person has avitaminosis A, regeneration is slower than normal and not complete at the time. Several hours of night driving could easily decrease the acuity of sight of a vitamin A deficient person to the point where his chance of having an accident would be greatly increased.

Being an individual affair, automobile driving at night does not enable one to compare his skill directly with that of a person with normal dark adaptation. Hence, many persons probably accept minor degrees of difficulty in night driving as the usual experience of all drivers.

This was illustrated by the experience that the writer, whose photometer reading was normal, had of going from bright daylight into a darkened cinema along with a person that, due to the faint illumination, had more difficulty in reaching a seat than himself. Vitamin A deficiency was predicted for this case, and a low reading was obtained on the visual photometer. After carotene medication, an improvement in activities in faint illumination under similar circumstances, as well as in the photometer reading, was noted. Similar experiences in a darkened cinema were noted by several other people with low photometric readings, and this is an interesting demonstration that mild degrees of hemeralopia are accepted as the normal situation, unless skill in performing an act in diminished illumination is compared with the simultaneous performance of a similar act by a person with normal dark adaptation.

Mason[22] has recently reviewed the minimal visual requirements for safe automobile driving, as required by forty-seven of the states in this country. No consideration was given hemeralopia, nor was any test mentioned that would detect it.

As defined by Edmund,[2] nyctalopia signifies the condition of being easily dazzled by light. In other words, a person with nyctalopia complains of being dazzled by an illumination that has no dazzling effect upon normal persons. While nyctalopia can be due to various eye diseases, Edmund has shown that it is present in some persons who have hemeralopia due to vitamin A deficiency. He expresses the belief that what is commonly known among autoists as "glare blindness", due to exposure to bright lights while driving at night, may be a manifestation of vitamin A deficiency.

Statistics show that the motor vehicle accident rate on a mileage basis is much higher at night than in the daytime. The National Safety Council,[23] from data collected in a typical eastern state, Connecticut, in 1933, was able to show that accidents and fatalities due to automobiles reached a peak at dusk and for the night hours maintained a high level in spite of the fact that the traffic curve declined rapidly. The presence of hemeralopia in many drivers, along with its accompanying drop in efficiency of vision for dim illumination, is one possible explanation that has been overlooked by traffic experts in explaining the greater hazards of night driving.

One can safely conclude that a person with hemeralopia that drives a car at night stands a greater chance of having an accident than he would if his vision for dim illumination were normal. The demonstration, by means of the photometer tests, that hemeralopia due to vitamin A deficiency is common in adults gives considerable importance to this problem. The extent and true significance of this fact can be established only by extensive field surveys made by groups interested in traffic problems.

Vitamin A therapy offers a simple cure for practically all persons that are found to have hemeralopia due to avitaminosis A. This, of course, is in marked contrast to many other visual defects that are liable to interfere with proper automobile driving.

If normal vision in dim illumination is important to a driver of an automobile, it is certainly more important to those engaged in piloting aircraft.

Berens and Smith,[24] in 1931, pointed out that a test of adaptation to light and visual acuity in reduced illumination was not included among the eye examinations required of aviators in the United States, although it was included in the "International Visual Standards for Aviators" as adopted by certain foreign countries. Whitehead[25] has recently listed the revised physical standards for aircraft pilots, as set forth by the United States Bureau of Air Commerce. "The following tests are made on the eyes—visual acuity, depth perception, ocular muscle balance, accommodation, central color vision (test for color blindness), visual fields, inspection and ophthalmoscopic examination". *No mention is made of a test for adaptation to light and visual acuity in reduced illumination.* The only consideration given to night blindness is the statement that retinitis pigmentosa, which can cause it, disqualifies a candidate. Wright,[26] in comparing the requirements of the Bureau of Air Commerce and the military aviation medical examinations, does not mention any test for hemeralopia. Since the tests for visual acuity and visual fields are performed in good illumination, there is no procedure on this list that will detect essential hemeralopia. It has been amply demonstrated that normal visual acuity, as usually tested, is no guarantee against the presence of night blindness. Frandsen[1] had many hemeralopes with 20/20 day vision, who had very poor vision after dark. The efficiency of the 20/20 vision for daylight, as required of commercial and military pilots, would certainly be decreased by even a minor degree of hemeralopia when the person is forced to perform in dim illumination.

A review of the *Quarterly Cumulative Index Medicus* for the past ten years did not reveal a single paper with a title relating to night blindness in aviation. A consideration of this problem by medical aviation experts would be well worth the effort in view of the recent investigations showing the prevalence of essential hemeralopia in this country and the fact that the eye examinations now commonly used will not detect this condition.

Besides these two aspects, automobiles and aircraft, night blindness is certainly of importance in the railroad field. There are many additional trades and industries in which efficient night sight is important. In our own profession, the roentgenologist, who does fluoroscopies in a darkened room, is an outstanding example. Certainly every fluoroscopist should have his visual acuity for dim illumination tested by means of a visual photometer.

SUMMARY

Personal observations and data obtained from the literature show that night blindness due to vitamin A deficiency decreases the efficiency of automobile driving at night and increases the probability of an accident. In view of the reported prevalence of hemeralopia, it is suggested that this problem is of sufficient practical importance to warrant an extensive study by traffic experts. It has been pointed out that at the present time no test for the presence of essential hemeralopia is required for either automobile drivers or aviators.

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NOTE: This material was presented to the members of the Massachusetts Safety Council and to Dr. H. R. deSilva of the Harvard Traffic School, who feel that the subject deserves further study. Dr. deSilva, in an extensive statistical and psychologic study of both automobile accidents and drivers, has found that from 5 to 10 per cent of drivers have marked difficulty in driving at night or are subject to severe degrees of glare blindness. Many of these people have given up night driving. In collaboration with Dr. deSilva a large series of persons involved in automobile accidents at night or complaining of difficulties in night driving are at present being studied to determine whether vitamin A deficiency plays a part.

MEDICAL PROGRESS

PROGRESS IN NEUROLOGY IN 1935

BY JULIUS LOMAN, M.D.*

Introduction

PROBABLY the most important advances in neurology in 1935 were made along the following three directions. First, increasing data through experimental and clinical methods are gradually giving neurologists more and more definite understanding of the autonomic functions centered in both the cerebral cortex and hypothalamus. Secondly, the technic of electroencephalography, that is, the recording of electrical waves coming off the brain, has received a wide application both here and abroad. Many interesting and important observations have been made by the use of this fascinating method of studying brain activity. Thirdly, concerning the clinical syndromes, definite advance has been made in the understanding of the chemistry and neurogenesis of myasthenia gravis, an understanding largely made possible by the very important discoveries of Loewi and Dale, who have demonstrated the intimate relation between the chemistry of the autonomic nervous system and muscle function.

PHYSIOLOGIC AND PATHOLOGICAL STUDIES

Electroencephalography

Berger[1] reports some of his many observations in electroencephalography gathered over a period of several years. By means of a highly sensitive galvanometer connected to the patient's scalp by means of a needle or pad electrodes and to an oscillograph, he has obtained electrical records from the brain under various physiologic and pathologic conditions. Electroencephalograms are best obtained under conditions of complete physical and mental relaxation. Fear, pain and anxiety change the curves. In newborn children, waves seen in the adult are absent. Chloroform causes the chief oscillations, 10 per second, to disappear. Brain-stem narcotics give rise to an exaggeratedly rapid course and incomplete formation of the characteristic oscillations. Berger believes that the characteristic oscillations arise from the cerebral cortex. His experiments with narcotics suggest, in regard to the origin of unconsciousness, that either the cortical activity may be extinguished or the sudden removal of inhibition from the cerebral cortex may suspend consciousness. Adrian and Matthews[2] maintain that the Berger or alpha waves arise from the occipital cortex, being discharged from the neurones concerned with pattern vision. These characteristic waves are at their maximum when the eyes are closed, when the mind is at rest, and when all external stimuli are reduced to a minimum. They are abolished upon opening the eyes, by mental effort and by sensory excitation to which "affect" is attached. It is interesting to note that even when the subject is in complete darkness, opening and closing the eyes produce the above-mentioned effects. The rhythm is seen to disappear during intellectual exercise and to reappear when the attention wanders.

Very interesting data have been gathered by Gibbs, Davis and Lennox[3] in electroencephalographic studies in conditions of impaired consciousness, including sleep, unconsciousness from breathing nitrogen, unconsciousness from failure of the cerebral blood supply and clouded consciousness from overventilation. These authors found that in sleep the frequency of the Berger waves (10 to 20 per second) decreases to between 1 and 5 per second. Very characteristic waves appear in petit mal attacks—groups of large, slow waves having a frequency of 3 per second and the appearance of an "egg and dart design". In grand mal attacks the waves increase in amplitude, and the convulsion begins. As the clonic state sets in, the first waves tend to clump together into slower waves of 5 per second. As the convulsive movements die down, the amplitude and frequency of waves decrease so that during the posteonvulsive stupor the record is nearly flat. During unconsciousness from nitrogen breath-

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ing, cerebral anemia and overventilation of the lungs, the frequency of the waves decreases to between 1 and 5 per second.

Visceral function in the brain

The higher centers and the pathways of the autonomic nervous system are discussed by Vonderahe.[4] The hypothalamus he designates as that anatomic division which forms the basis of inquiry as to its relation to visceral function. These functions have to do with the regulation of fat, protein, carbohydrate, mineral and water metabolism, also with temperature regulation, regulation of internal and external glandular secretion, the vasomotor mechanism and sleep. According to recent work, the anterior portion of the hypothalamus is related to the parasympathetic function and the posterior portion to sympathetic function. Clinical syndromes relating to each portion of the hypothalamus or to both portions are discussed by the author.

Watts[5] describes interesting experiments on the results of faradic stimulation of the cortex. Stimulation of the premotor area causes vigorous peristalsis of the small intestine, in some instances with the production of intussusception, sometimes multiple. On the other hand, stimulation of the motor area has no effect on intestinal movements. Watts describes a case in which the first symptom was a ravenous appetite, as a result of a proved frontal tumor.

Fulton[6] believes, in view of the abnormal peristalsis resulting from frontal lesions, that symptoms of marked hunger may be due to increased motility of the stomach. Globus[7] cites several cases illustrating association of vegetative function with cerebral lesions situated in or about the hypothalamus. The symptoms presented in these cases include disturbances in water balance, fat metabolism, in primary and secondary sex characters, in heat regulation, in intestinal peristalsis and in appetite.

Upper motor neuron lesions

Fulton and Viets[8] analyze the differences between paralytic syndromes produced by lesions in the premotor area and pyramidal tracts. The authors point out the following distinctions, based on studies of monkeys, apes and man. When the lesion is restricted to the pyramidal tracts or to their cells of origin, there result a flaccid paralysis, muscle atrophy, transient depression of all reflexes and the presence of Babinski or Chaddock signs. Complete destruction causes permanent Babinski or Chaddock, but the paralysis, flaccidity and reflex changes tend with time to disappear. Lesions of the premotor projection area of the cortex, which also mediate voluntary movements, result in spastic paralysis, disturbance in skilled movements, forced grasping, vasomotor disturbance, increased deep reflexes and the signs of Rossolimo, Mendel-Bechterew and Hoffmann. Forced grasping and vasomotor disturbances are transient, but disturbances in skilled movements and the spinal reflex signs tend to persist.

Hemiplegia in man is generally produced by the combined destruction of motor and premotor components of the upper motor neuron, thus accounting for the combination of signs frequently seen and the more severe, lasting paralysis than when only the pyramidal tracts are involved.

TRAUMA TO THE BRAIN AND SPECIAL THERAPEUTIC PROCEDURES

Subdural hematoma

Although probably the most difficult of traumatic types of cerebral hemorrhage to diagnose, subdural hematoma is being more and more readily recognized. Gardner[9] reports 22 cases in which the following points are of interest: the blow to the skull was usually mild; the average interval between the trauma and operation or necropsy was 12½ weeks, the shortest being 5 weeks and the longest 33 weeks; the most common symptoms were headache, altered intellect and somnolence that was out of all proportion to the degree of intracranial pressure, the most stuporous patient often having a normal or even a subnormal pressure. In only 8 cases did the neurologic signs indicate the site of the lesion. In 5 of the cases the signs were misleading. Of the 20 cases operated upon, there were 90 per cent recoveries and 70 per cent cures, showing that subdural hematoma when recognized and properly treated is one of the most benign of all expanding intracranial lesions.

Many of these features of subdural hematoma are corroborated by Grant,[10] who reports a series of 16 cases of chronic subdural hematoma, verified at operation or at autopsy. There were 5 deaths, 4 of which were postoperative. Grant also found that slight cranial injury can produce a hematoma and that neurologic evidence of its presence may be extremely vague and misleading, although headache and lethargy are always pronounced. Because of uncertainty of localization and because of the large percentage of cases in which bilateral hematoma occurs, the importance of bilateral operation is stressed by Grant.

Follow-up of skull fractures

Of 100 cases of skull fracture occurring 5 to 13 years previously, Early[11] found that 63 were completely well, 31 had residual symptoms, but were able to maintain a reasonably normal life, and 6 were incapacitated. Headache persisted in the 37 practically cured cases, vertigo was still present in 15, and 7 had defective memory; 3 developed a traumatic psychosis and 4 personality changes. Petit mal convulsions developed

in 3 cases, and 2 had generalized convulsions. Three patients showed weakness of one side of the body. Twenty-six of the cases were operated upon. Of these, 14 had a complete recovery, 10 a partial recovery and 2 were incapacitated. Of the 72 that received conservative treatment, 49 completely recovered, 22 partially recovered and 3 were incapacitated. In cases receiving conservative treatment, Early recommends prompt and continued reduction of increased intracranial pressure to normal, bed rest in a hospital for at least 3 weeks and a long period of physical and mental rest after discharge.

Therapeutic pneumocranium

Drayton[12] obtained satisfactory results in most cases of head injury complaining of headache, dizziness, changes in character and convulsions. Of a group of 46 complaining of headache and dizziness, 41 were completely relieved of the former symptom and 43 of the latter symptom. Of a group of 10 having convulsions, 2 were completely relieved and 6 improved. Of a group of 43 showing changes in character, 21 became normally adjusted and 16 were improved. Drayton recommends a repetition of insufflation two or even three times if satisfactory results are not obtained following the first attempt.

Colloidal thorium dioxide in cerebral diagnosis

Further use of this radiopaque substance has been made in ventriculography by Freeman, Schoenfeld and Moore[13] and by Twining and Rowbotham.[14] The former authors used the following technic for localizing such lesions as neoplasms and abscesses. After withdrawing cerebrospinal fluid from the posterior horns of the lateral ventricles, they inject 3 cc. of thorium dioxide; x-ray films are immediately taken. These authors have found that the substance is eliminated from the body in about 4 hours. It causes some irritation, as shown by initial leukocytosis followed by a lymphocytic and particularly a histiocytic response. The thorium dioxide is freely miscible with the cerebral fluid, permitting complete visualization of the ventricular system, including the third ventricle, aqueduct and the fourth ventricle. The danger in the use of this substance seems to be in ventricular obstruction.

Twining and Rowbotham[14] found thorium dioxide most suitable as a contrast medium for ventriculography. Headache did not follow its use, as with injections of air. The authors inject 10 cc. of thorium dioxide into the ventricle. They point out the difficulties in interpreting the radiograms.

Loman and Myerson[15] describe a technic for directly injecting thorium dioxide into the carotid artery for visualization of the cerebral vessels. By its use the disadvantages of cutting down and exposing the vessel are eliminated. In this method an apparatus recording the carotid pressure is used in order to make certain that a perfect puncture has been made. Rapidity of injection of the thorium dioxide (usually 10 cc.) and slowing of the cerebral circulation by manual compression are requisites for good radiograms. Excellent arteriograms are obtained with injection of 10 cc. of this substance. For the outline of the cerebral veins 15 to 20 cc. of thorium dioxide are necessary, x-rays being taken at 2 to 3 second intervals following the taking of the arteriogram. In 35 cases injected by the method no untoward effects have been noted, either acute or chronic. The cases have been followed for more than 2 years.

The use of sucrose for diminishing cerebrospinal fluid pressure

Bullock, Gregersen and Kinney[16] find that sucrose is definitely superior to glucose or sodium chloride in diminishing cerebrospinal fluid pressure. Their experiments were done on dogs, anesthetized with sodium amytal. Three to 8 grams of sucrose per kilogram body weight, injected intravenously in a 50 per cent solution, reduced the pressure for 5 to 8 hours, the reduction depending upon the height of the initial pressure. By contrast, 50 per cent glucose and 30 per cent sodium chloride solution reduced the pressure for only 2 to 3 hours. Furthermore, the fall in pressure created by these latter two solutions was followed by a secondary increase in pressure. Injections of sucrose, however, were not followed by a secondary rise in pressure except in 3 cases. The rise in pressure in these latter cases was attributed to other causes.

Gregersen and Wright[17] demonstrate that the secondary rise in cerebrospinal fluid pressure, following injections of hypertonic solutions of glucose, is due to the fact that the sugar diffuses into the spinal fluid and makes it hypertonic, so that when the sugar in the blood stream falls, the brain remains hypertonic and becomes edematous.

Postpuncture headache

Reese and Shulak[18] have used the following procedure for the prevention of this type of headache. Five to 10 minutes before lumbar puncture is done, 50 cc. of a 0.225 per cent saline solution are given intravenously. Following the puncture, the patient is kept quiet for 1 hour and then allowed to get up. By the use of this technic, headache was eliminated in a group of 25 patients. When patients that had already developed headache following lumbar puncture were given the hypotonic solution, there were varying degrees of response. In some of these cases the headache disappeared in ap-

proximately 30 minutes after the injection of the hypotonic solution. In other cases there were varying degrees of improvement.

MULTIPLE SCLEROSIS

Pathogenesis

Probably the most important recent experimental work done on this disease is that by Putnam.[19] By means of a special technic this author produced obstruction of the cerebral veins in dogs. The animals were later killed by bleeding, and their brains examined. Of 14 successful operations, all but 2 showed cerebral lesions. These lesions varied, depending upon the agents used to produce the obstruction and the length of time the animals were allowed to survive. Early lesions closely resembled those of nonsuppurative encephalitis. Later stages of the lesions showed plaques of demyelination with practically complete preservation of the axis cylinders and with dense fibrous gliosis confined to the white matter. Such lesions are so similar to those seen in multiple sclerosis that the author concludes that venular obstruction is the essential immediate antecedent of the formation of sclerotic plaques.

Lead in multiple sclerosis

The theory advanced by Cone, Russell and Hardwood that lead is implicated in the production of multiple sclerosis has not been substantiated, according to the work of Boshes.[20] Of 16 cases of multiple sclerosis, in which the spinal fluid was examined, only 1 showed a positive result. This patient had previously received sodium iodide, which normally increases the output of lead in the excreta. The spinal fluid was also examined in other abnormal conditions, including a case of lead poisoning. Of these, only the latter case showed abundant lead crystals in the spinal fluid.

Therapy in multiple sclerosis

Brickner[21] has found quinine to be definitely beneficial in the treatment of multiple sclerosis. Of 49 patients treated for a long time, 25 showed remissions or were so improved as to be able to work. None who were able to work before treatment are now unable to do so. Ten cases became apparently arrested. Five are worse than before treatment, and 3 have died. Brickner recommends the administration of quinine indefinitely, avoiding cinchonism. Best results are expected when the drug is given early in the disease and particularly in cases showing slight progress.

ANTERIOR POLIOMYELITIS

Symptomatology and treatment

Neal[22] emphasizes the changed conception of poliomyelitis. The disease is now believed to involve the central nervous system from the beginning. The incubation period is probably between 6 and 18 days. Nonparalytic and paralytic cases may follow equally severe symptoms. There is no characteristic fever curve. A disproportionate pulse rate may mean early bulbar involvement. Generalized hyperesthesia may be an early symptom. Drowsiness, alternating with irritability, is characteristic. Delirium and convulsions are rare. Stiffness of the neck is not so marked as in meningitis. The deep reflexes are exaggerated in the early stage, later becoming unequal, diminished or lost, especially when paralysis is about to develop. Paralysis appears commonly on the second or third day, if at all, except in dromedary types. The spinal fluid in most cases is increased in amount, in cells and slightly in protein. Rarely is it completely normal.

As regards treatment, it has been shown in animal experimentation that convalescent serum cannot be used sufficiently early to prevent invasion of the cord. Lumbar puncture increases the comfort of the patient. In cases of respiratory embarrassment, the patient should be placed in a suitable respirator. Neal is inclined to believe that the seriousness of poliomyelitis is overestimated in view of the large percentage of nonparalytic cases and of the many paralyzed cases that completely or almost completely recover under proper treatment.

Brodie,[23] like Neal, believes that serum is of no benefit in poliomyelitis, for the first stage is probably by direct nerve transmission rather than by the hematogenous or cerebrospinal fluid route. This causes involvement of the midbrain, hypothalamus and perhaps the brain-stem, after which the virus seems to halt for a time before progressing to the spinal cord. The serum seems to have difficulty in reaching and penetrating the central nervous system, which is well insulated from the hematogenous system. Furthermore, if the serum should reach the central nervous system, it could not dislodge the virus, which appears to be fixed to the nerve cells, nor could it prevent further propagation, for a virus will grow on tissue culture in the presence of its own antibody. Brodie found that, in monkeys infected with anterior poliomyelitis virus, doses of serum much larger and given earlier than are usually given to patients fail to affect the clinical course of the disease.

Schultz and Gebhardt[24] point out, as a result of their experiments on the protective value of immune serum, that the serum cannot be depended upon to modify infection with poliomyelitis and that the only hope of controlling the disease lies in the possibility of active immunization.

Kolmer, Klugh and Rule[25] have administered poliomyelitis virus (prepared from remote passage virus and attenuated by sodium ricinoleate)

to a selected group of 25 children, varying in age from 8 months to 15 years. Fifteen of these cases showed no antibody, as measured by serum neutralization, before immunization. Eleven of these showed large amounts of antibody 1 week after the last dose of vaccine. In 10 that had shown the presence of antiviral antibody before immunization there developed a considerable increase in antibody after vaccination. Mild local reactions at the sites of subcutaneous injection, with a slight elevation of temperature and slight leukocytosis, subsided within 48 hours.

MENINGITIS

Fresh human serum in meningococcus meningitis

Fondé[26] has found that fresh human serum given intraspinally is a valuable adjunct to commercial serum in the treatment of meningococcus meningitis. Fifteen cc. of fresh serum were given in 2 cases, preceded and followed by antiserum. In 1, the spinal fluid became sterile 48 hours after the injection. In the other patient, who was critically ill and who received the fresh human serum 24 hours after the initial administration of the antiserum, convalescence and recovery occurred rapidly. The value of the serum is corroborated by others.

Tuberculous meningitis

Walker[27] discusses the symptomatology and pathology of tuberculous meningitis in adults, after studying 45 hospital cases. It was found that headache is a universal symptom. Common symptoms in early cases are backache, vomiting, constipation, depression, moodiness, emotional disturbances, memory impairment and lack of concentration. Paranoid states, euphoria and speech disturbances are sometimes present. Later, actual delirium, stupor and coma, convulsions, bulbar disturbances, ocular palsies and various types of paralyses usher in the end.

Walker distinguishes three stages in the pathological development of the disease. First, there is tubercle formation in the absence of clinical symptoms. The second stage begins with subpial infection when the broken-down material escapes into the cerebrospinal fluid with the production of neurologic symptoms, particularly headache and mental disturbances. The third stage is the production of massive meningeal infection with exudate and local softening. Recovery, which sometimes occurs, takes place before leakage or too rapid escape of the bacilli into the subarachnoid space has occurred. Treatment by early and frequent drainage is very important because it removes many bacilli and their products.

NEUROSYPHILIS

Blood Wassermann in neurosyphilis

The importance of examining the spinal fluid in every case of syphilis is shown in a study by Menninger and Bromberg.[28] In 500 cases of various types of neurosyphilis, the blood Wassermann was negative, in 297 cases of asymptomatic neurolues, in 101 patients or 33.5 per cent; in 133 cases of tabes, in 37 patients or 27.8 per cent; in 38 cases of general paresis, in 8 patients or 21.1 per cent; in 19 cases of taboparesis, in 5 patients or 26.3 per cent; in 13 cases of meningovascular lues, in 4 patients or 30.8 per cent.

Treatment of general paresis

Solomon and Epstein[29] have analyzed the results obtained by malarial and other methods of therapy in 173 patients suffering from general paresis. Good improvement was obtained in 36.4 per cent and definite improvement in 27.1 per cent. In 169 of the patients, the spinal fluid became completely normal in 36.7 per cent, greatly improved in 20.7 per cent and somewhat improved in 18.3 per cent. Early treatment with malaria in patients whose general condition is good will cause the disease to be arrested in most cases. In addition to the malaria treatment, various other types of therapy were given, particularly tryparsamide. It was found that combinations of other methods following malaria therapy give the most satisfactory results.

Life expectancy in general paresis

Derby[30] finds that the duration of life of paretics admitted to the Brooklyn State Hospital over a period of 11 years was from 1 year and 6 months to nearly 2 years and 9 months. In those that lived long enough for malarial therapy to have taken effect, the average duration of life increased in the same period from 2 years and 9 months to 3 years and 10 months.

Hemorrhagic encephalitis and myelitis following intravenous arsphenamine

Glaser, Imerman and Imerman[31] have analyzed the records of 158 patients that manifested nervous system complications following arsphenamine injection. They point out that the combined statistics collected from the literature on 191,125 patients, who had received 1,108,778 injections of arsphenamine compounds, showed 1,377 complications, or 1 occurring in every 129 cases, and 1 death in every 2,699 cases. In the group they analyzed, there were 146 cases of encephalitis, 8 of myelitis and 4 of encephalomyelitis. The mortality was 76 per cent. In 50 per cent of the cases, hemorrhagic encephalitis followed the second injection. In about 20 per cent, death occurred within the first 24 hours. General symptoms of headache, vomiting, chills and dizziness were present. Of 146 patients, 80 per cent had convulsions. Involvement of the III, IV, VI and VIII cranial nerves

was also noted. Various types of reflex changes occurred in 30 per cent. Mental disturbances, consisting of delirium and disorientation, occurred in less than 50 per cent. Of 39 patients, 63 per cent had increased spinal fluid pressure. Subarachnoid hemorrhage was present in 26 per cent. Many cases with brain and cord disturbances gave clinical signs of meningitis.

The following prophylactic measures are recommended: (1) arsphenamines should not be given to patients with damaged kidneys; (2) best care and technic should be used in administering the drug; (3) ephedrin sulfate (50 mg.) should be given for 2 days prior to injection; (4) adrenalin (1 cc. of 1:1000 solution, intramuscularly) 10 minutes before the injection is recommended; (5) atropin sulfate (1/150 grain, hypodermically) 15 minutes before the arsphenamine injection is also advised. Curative measures for the toxic phenomena are as follows: (1) adrenalin chloride, 1 cc. (1:1000) intramuscularly, followed by 1 cc. (1:100,000) intravenously; (2) sodium thiosulfate; (3) sodium dehydrocholate; (4) calcium thiosulfate; (5) calcium chloride; (6) ephedrin sulfate; (7) 50 per cent glucose solution.

Acute syphilitic meningitis

Merritt and Moore[32] have analyzed 80 cases of this type of luetic infection treated at the Boston City Hospital or admitted to the Massachusetts General, Peter Bent Brigham or Boston Psychopathic Hospital during the past 15 years. Their studies show that less than 5 per cent of syphilis appears in this form. It usually occurs in young adults. The meningeal symptoms usually develop in the first year of the primary infection but may develop at any time after the initial lesion.

Clinically, the cases may be divided into three groups: (1) acute syphilitic hydrocephalus, in which headache, nausea, vomiting, choked discs and meningeal signs are present, indicating involvement of the meninges in the posterior fossa that interferes with the circulation of the cerebrospinal fluid and thus causes the development of acute hydrocephalus; (2) acute vertical meningitis, in which headache, nausea and vomiting, or convulsions and mental symptoms indicating involvement over the vertex of the brain are present; (3) acute basilar meningitis, in which cranial nerve palsies indicating meningitis at the base of the brain are prominent.

The blood Wassermann was found to be positive in only 60 per cent of the cases. The diagnosis of this form of meningitis can be immediately established only by a lumbar puncture. The spinal fluid pressure is usually increased, at times being as high as 600 to 700 mm. of water. There is also found a moderate to marked pleocytosis, ranging from 25 to 2000 cells per cubic millimeter, with lymphocytes predominating; protein is also increased. The glucose is either normal or decreased, ranging from 18 to 84 milligrams per 100 cc. Chlorides are usually normal or slightly diminished, ranging from 636 to 742 milligrams per 100 cc. The colloidal gold curve may be of any type. The spinal fluid Wassermann was found to be positive in approximately 90 per cent. Spirochetes are rarely found in the spinal fluid.

The treatment recommended for acute syphilitic meningitis is the use of intravenous injections of arsphenamine or their derivatives and frequent lumbar punctures. The acute symptoms rapidly subside with treatment. The ultimate prognosis is good, when proper follow-up treatment is given.

SPINAL CORD

The value of liver therapy in combined system disease due to pernicious anemia

Strauss, Solomon, Schneider and Patek, Jr.[33] point out that the disagreements in the literature on the value of liver in the neurologic disturbances of pernicious anemia are due to the lack of standardization of the doses of liver and to the recording of the results of treatment for too short a period of time. The authors followed 26 patients complaining either of spasticity or ataxia, or both, with definite locomotor disturbances, paresthesias, disturbances in vibratory sense and muscle weakness for a period of from 14 to 46 months. Complete arrest occurred in every case; in some instances, previously bedridden patients were able to return to their former occupations. The authors emphasize that gains in strength and activity are not necessarily evidence of regeneration of the spinal cord fibers or cells. This latter point of view has previously been expressed by Grinker.

Farquharson[34] also obtained encouraging results with large doses of potent liver preparations combined with prolonged bed rest for a period of at least 3 months. None of those that followed the treatment faithfully became worse and many showed striking improvement.

Roentgen therapy in syringomyelia

O'Brien[35] gives his results in 30 cases of syringomyelia treated by x-ray. Nineteen cases showed definite improvement, either subjective or objective, or both. The improvement consisted in the return of thermal sensation, cessation of pain, restitution of voluntary muscular movements of the legs and arms, disappearance of ataxia, restoration of normal speech and deglutition, and healing of trophic ulcers. Some cases were able to return to gainful occupations. The age of the patient or the duration of the disease did not appear to militate against beneficial symptomatic relief. Favorable results occurred from the use of medium as well as high voltage x-rays.

CRANIAL AND PERIPHERAL NERVES

Trigeminal neuralgia

For this painful affliction Grant[36] recommends only two methods of treatment: (1) alcohol injections into any of the three branches of the trigeminal nerve and (2) section of the central root behind the ganglion. In indefinite cases, the affected branch can be determined by novocaine injections before proceeding with the alcohol. This technic prevents many errors. Permanent relief is obtained by section of the sensory root, either total or subtotal. Only 1 death in 100 such operations performed by Grant and 2 cases of facial palsy occurred. Grant also recommends section of the sensory root in pain due to facial cancers. Of 35 such cases, 26 showed complete relief and 6 marked improvement; 2 fatalities resulted.

Vitamin deficiency and nervous system pathology

The relation of vitamin B deficiency to lesions of the nervous system, especially of the spinal cord, such as are seen in pernicious anemia, pellagra and alcoholism, has been studied by Gildea, Castle, Gildea and Cobb.[37] Two series of experiments were carried out. In the first series, 17 dogs given a diet deficient in vitamin B developed acute disturbances of the central nervous system and died without having been treated with vitamin B concentrates. The nervous system of these animals showed only minimal histologic lesions. In the second series, 8 dogs, on a similar diet, whose acute neurologic signs were repeatedly and temporarily relieved with vitamin B concentrates, gradually developed a residual degree of spastic ataxia and, eventually, motor paralysis with reflexes present. In all but 1 of these animals, definite lesions of the central nervous system were found. These changes included evidences of degeneration in the ventral horn cells, loss of myelin of the spinal cord and fatty changes in the peripheral nerves. Only slight pathologic changes were found in the cerebral cortex. The authors conclude that prolonged dietary deficiency is necessary before demyelination and other lesions of the central nervous system are produced.

In a study on the etiology of alcoholic polyneuritis, Strauss[38] found that in 10 patients the neuritis improved when they were kept on a well balanced, high vitamin diet, supplemented by yeast and its products and including parenteral administration of vitamin B and liver extract, even though a pint to a quart of whiskey was allowed daily. Patients that were treated without whiskey showed no difference in the course of their polyneuritis, if given a good diet.

EPILEPSY

Cortical autonomic epilepsy

Watts and Frazier[39] cite 2 interesting cases of epilepsy in which such symptoms as nausea, epigastric distress and vomiting appeared to abort or were substituted for convulsions. Thus, it would appear that a neuronal discharge in epilepsy may manifest itself either through the gastrointestinal musculature or through the skeletal musculature. The authors point out that epigastric aura and other visceral sensations described by epileptics are not referred pains but are due to vigorous and perhaps abnormal movements of the intestinal tract. As a result of their observations, Watts and Frazier postulate a motor autonomic representation for the gastrointestinal tract in the cortex of man. Such a postulation is in line with the observations of Penfield and Gage and of Watts and Fulton.

Epilepsy following head injury

Glaser and Shafer[40] have reviewed a large number of case reports, including 1 of their own, in an attempt to determine the relation between head injury and convulsions. The following salient features are brought out. Epilepsy occurs with sufficient frequency following head injury to be of clinical importance. For early convulsions to occur, the brain injury must have been severe. Convulsions usually develop from 6 months to 2 years following injury, less frequently from 2 to 7 years afterward and infrequently from 7 to 20 years afterward. Head injury sensitizes the brain so that extraneous circumstances may more readily bring on convulsions. There is a wide discrepancy in the literature concerning the incidence of generalized convulsions following head trauma.

NARCOLEPSY

Therapy

Prinzmetal and Bloomberg[41] have shown that benzedrine (phenylisopropylamine) is definitely more effective in the treatment of narcolepsy than is ephedrin. Well-controlled studies, in which both the above drugs were administered to 9 patients, were carried out. The dose of benzedrine used varied from 10 milligrams once daily to 40 milligrams three times daily, depending on the age and the severity of the case. In every case complete relief from attacks of sleep and practically complete relief from cataplexy resulted when suitable doses were given. The drug was found to be about three times as effective as ephedrin. No diminution in the effectiveness of the drug was observed over long periods. Insomnia, restlessness and hyperexcitability, symptoms that may occur from large doses of benzedrine, were controlled by reducing the dosage.

MIGRAINE

For the past three years Lennox and von Storch[42] have treated 120 patients suffering from migraine. Of these 89 per cent experienced abrupt and complete cessation of the headache with the initial use of ergotamin. The beneficial action of the drug appeared to be specific or nearly specific in the migrainous type of headache, for only 7 out of 45 patients with headache from other causes were definitely improved after the intravenous injection of the drug. In general, patients with migraine headache responded to subsequent injections as they did to the initial injection. The authors found that the headache once aborted tends to recur at shorter intervals, especially in the first few weeks or months of treatment. Unpleasant symptoms following injection included nausea and vomiting in a large percentage of the cases. Occasionally gastrointestinal symptoms occurred. Lennox and von Storch have seen no permanent or serious ill-effects from the use of the drug. Caution in administering it is advised to patients with arterial disease, since it raises the blood pressure. Excessive and long-continued use carries the danger of ergotism.

MYASTHENIA GRAVIS

Boothby[43] states that of 65 patients with myasthenia gravis seen at the Mayo Clinic over a period of 3 years 13 have died, 15 are now able to carry on practically full-time work and are self-supporting, 25 are able to do small chores and 5 remain unimproved. Glycine was not found to be specific for the fatigability of myasthenia gravis, but had a more favorable effect on a larger group of cases of nonspecific fatigability.

On the basis that potassium ions enhance the stimulating effect of acetylcholine on the motor nerve endings, thereby increasing muscle contraction, Laurent and Walther[44] administered potassium chloride in doses of 2 grams orally five times a day to 3 myasthenia gravis patients. Subjective improvement starting one-half hour after each dose and passing off in the course of the next half hour was noted. Objectively, there was slight diminution in ptosis of the lids, a little improvement in the range of ocular movements and increases in from 3 to 5 pounds in the power of the handgrasp. In other cases, a dose of 12 grams was given. In these patients, ptosis of the lids almost completely disappeared 45 minutes afterward.

Pritchard[45] has treated 7 patients suffering from myasthenia gravis with injections of prostigmin and atropin sulfate. Improvement in some measure resulted within the first 5 minutes after the injections and reached its height in about 30 minutes, passing off almost completely in 8 hours. These results confirm those of Walker, published in 1934.

Viets and Schwab[46] report 12 cases of myasthenia gravis, in which injections of prostigmin caused a marked, but temporary, remission of muscular weakness. In addition to its therapeutic use, these authors recommend the drug as a diagnostic test for the disease.

Electromyographic studies of myasthenia gravis carried out by Lindsley[47] show that the effect on the muscles gives abnormally marked fluctuations in amplitude but that the nerve functions normally. Following intramuscular injection of prostigmin, the action potentials become practically normal. These findings, Lindsley concludes, strongly suggest that acetylcholine, or a related substance, is liberated at the motor nerve endings and takes part in the transmission of nerve impulses to voluntary muscles.

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SCIENTIFIC EVENTS FOR THE YEAR

Among the notable achievements in the medical sciences for 1936 listed by *Science Service* are the following:

Detection of radium poisoning became more accurate through the application of a new type of screen-cathode quantum counter developed by Dr. Robley D. Evans, Massachusetts Institute of Technology, and said to be 10 to 100 times more sensitive than older methods of detecting radium in the body.

A new technic for investigating living matter by obtaining characteristic electrical patterns specific for each species and which, in animals, has already shown characteristic changes in the pattern on the advent of cancer and during ovulation and other physiologic processes was devised by Drs. Harold S. Burr, Cecil T. Lane and Leslie F. Nims, of Yale University.

Neutron rays are more effective than x-rays in killing tumors in animals and may prove more potent in destruction of cancers in human beings, Dr. John H. Lawrence, of Yale University, and his brother, Professor Ernest O. Lawrence, of the University of California, reported, on the basis of studies with the atom-smashing cyclotron invented by Professor Ernest O. Lawrence.

Further evidence that the adrenal and pituitary glands play an important role in diabetes was obtained by Dr. C. N. H. Long, of Yale University.

Influenza can be more widely spread through the air than was previously supposed, but ultraviolet light kills the influenza virus suspended in droplets in the air, studies by Drs. W. F. Wells and H. W. Brown, Harvard School of Public Health, indicated.

Successful use of placental extract in the treatment of hemophilia was reported by Drs. R. Cannon Eley and Charles F. McKhann, of Boston.

Epileptic seizures were predicted by electrical studies of the brain conducted by Drs. F. A. Gibbs and W. G. Lennox, of Harvard Medical School.

DO YOU KNOW?

First instruction in first aid was proposed by George Ryerson Fowler and given in 1885 at the annual encampment of the New York State militia at Peekskill, N. Y.

A Dream-Book 3,000 years old has been found which advised the ancient Egyptian what dreams were good and what ones were bad.

Philip Gibbs said: It is better to give than to lend, and it costs about the same.

Fatigue, excessive or unexplained, is a serious symptom. He who pushes himself to the point of true exhaustion is like a motorist who goes through a red light. He may do it once in safety, but in the end, the Fate which waits round the corner will get him.

Tuberculosis, heart disease and many other ailments, have fatigue as one of their symptoms. In fact, it is sometimes the earliest symptom, a warning to the wise to seek help early, when the chances for successful treatment are greatest.

More banking on curves of highways to avoid accidents is advocated by Joseph Barnett of the U. S. Bureau of Public Roads, after 900 tests at speeds up to 70 miles an hour.—*Excerpts from the Bulletin of the New York State Medical Society.*

CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTEMORTEM AND POSTMORTEM RECORDS AS USED IN WEEKLY CLINICOPATHOLOGICAL EXERCISES

FOUNDED BY RICHARD C. CABOT, M.D.

TRACY B. MALLORY, M.D., *Editor*

CASE 23021

PRESENTATION OF CASE

First Admission. A 54 year old Italian laborer was admitted complaining of weakness, headache and drowsiness.

Two weeks before entry the patient developed headache, most marked in the occipital region. At the same time he noticed weakness, fatigability and anorexia. He became constipated, felt rather feverish, but had no chills. There was profuse perspiration, and he felt sufficiently ill to go to bed for 5 days. He returned to work, however, but lassitude and weakness continued and he again was confined to bed. Shortly before entering the hospital he became quite drowsy, but there were no other symptoms of significance.

He had polyarthritis at the age of 33 years. For about 10 years he had tinnitus in his left ear and deafness in the right, associated with some vertigo.

Physical examination showed a well developed and nourished middle-aged man with slight distention of the neck veins. The patient lay flat in bed without discomfort. The pupils were equal and reacted normally to light. Fundal examination showed slight blurring of the nasal margins of the disks. The heart was slightly enlarged to the left. A presystolic rumble was audible in the apical region. A blowing systolic murmur was heard at both apex and base. The blood pressure was 110/70. There were numerous crackling, sticky rales and slight dulness at the bases posteriorly. Breath sounds and tactile fremitus were increased in intensity. Crackling rales and exaggerated breath sounds were audible in scattered small areas anteriorly. The spleen extended two fingerbreadths beneath the costal margin.

The temperature was 105°F., the pulse 100. The respirations were 28.

Examination of the urine showed a specific gravity of 1.018 with a slight trace of albumin. The remainder of the examination was negative. The blood showed a red cell count of 5,300,000, with a hemoglobin of 75 per cent. The white cell count was 14,300. A smear showed a differential count of 53 per cent polymorphonuclears, 10.5 lymphocytes, 4.0 young monocytes, 31.5 monocytes, 0.5 epithelioid cells and 0.5 eosinophils. Several sputum examinations were negative for tubercle bacilli. Repeated stool examinations were likewise negative. A Widal test was positive up to a dilution of 1:160. Agglutination reactions for paratyphoid and undulant fever were negative. Subsequently the Widal test became negative, and repeated blood cultures were negative. The sedimentation rate was 0.32 millimeters per minute. A lumbar puncture showed an initial pressure of 100; clear colorless fluid was removed and the dynamics were normal. A cell count showed 4 lymphocytes. There was no globulin. The sugar was 92 milligrams. A spinal fluid Wassermann was negative. The serum chlorides were equivalent to 106 cubic centimeters N/10 sodium chloride. The nonprotein nitrogen of the blood was 50 milligrams. Repeated urine, stool and bile cultures showed no pathogenic organisms. An electrocardiogram showed ectopic auricular tachycardia on one occasion and was otherwise negative.

X-ray examination of the chest was negative.

For the first two weeks in the hospital the patient's temperature fluctuated between 101° and 106.5°F. It showed an irregular diminution during the succeeding week and thereafter remained normal. During this febrile period he developed stiff neck, twitching and carphologia. Lumbar punctures showed the pressure to be within normal limits, but the cell count gradually rose to 100. All of the cells were lymphocytes. Spinal fluid protein and sugar were consistently within normal limits. After the temperature reached a normal level the central nervous system symptoms disappeared and his condition improved somewhat, although he had several attacks of paroxysmal tachycardia. Rales were audible at intervals at the bases posteriorly, but the remainder of the chest had cleared. X-ray examination shortly before discharge showed slight increase in the lung markings in the upper lung field, and there was questionable mottling. At this time it was noted that his red blood cell count had diminished to 3,500,000 and his hemoglobin to 55 per cent. He was given a transfusion and discharged improved on the fifty-third hospital day.

Final Admission (5 weeks later). Following his discharge the patient continued to improve in strength and well-being, and gained about 21 pounds. Six days before reentry, however, his face appeared to be flushed, and he had slight anorexia. On the following day there was increasing malaise, and he was definitely feverish. There was slight epistaxis on one occasion. Three days before entry he went to bed, and his temperature was found to be 104°F.

Subsequently he was very drowsy and occasionally slightly irrational. He had continuous hiccoughs and frequent eructations of gas. There was occasional cough with the production of a small amount of frothy sputum.

Physical examination showed a well developed and nourished man lying flat in bed sweating profusely but exhibiting no respiratory difficulty. The mucous membranes were rather dry. The pupils were small, slightly irregular and reacted sluggishly to light. The fundi were negative. The throat was injected, but there was no exudation or ulceration visible. Pea-sized inguinal nodes were palpable, but there was no other adenopathy. There was slight resistance to forward flexion of the neck but no definite stiffness. The heart was irregular in rhythm, and there was a pulse deficit at the wrist of about 40, with an apical rate of 120. The blood pressure was 110/60. A questionable mitral diastolic murmur was audible. There were crackling, moist rales and slightly increased breath sounds at the bases. The abdomen was soft, full and tympanitic. The liver edge extended about four fingers beneath the costal margin, and the spleen was enlarged two fingerbreadths. The remainder of the examination was negative.

The temperature was 105° F. The respirations were 30.

Examination of the urine was negative. The blood showed a red cell count of 6,600,000, with a hemoglobin of 70 per cent. The white cell count was 40,300. The smear showed 42 per cent polymorphonuclears, of which only 3 per cent were band forms, 8 lymphocytes, 42 monocytes, 1 eosinophils, 5 immature monocytes, and 2 erythroblasts. Stool examinations were negative. The nonprotein nitrogen of the blood was 61. A Hinton test was negative. Agglutination tests for undulant, typhoid and paratyphoid fevers were negative. A heterophil agglutination reaction was negative. Urine and stool cultures showed no pathogenic organisms. A lumbar puncture showed an initial pressure of 95 with normal dynamics. The total protein was 25, and the remainder of the examination was negative.

X-ray examination of the chest showed no change from that previously noted.

The temperature remained elevated between 102° and 106° F. The patient became irrational and developed definite stiffness of the neck and positive Kernigs. Another lumbar puncture was normal. He became progressively worse and died on the sixth hospital day, 3 months after his initial entry.

Differential Diagnosis

Dr. John H. Talbott: An explanation should be given at the start that I saw this patient during his first stay in the hospital and am not starting entirely from scratch, as is the usual custom at these exercises. I did not see him on the second admission, however, and do not know the results of the postmortem examination.

There are three or four facts that should be included in the history and I think it is permissible to mention them at this time. The present illness was more than 2 weeks in duration and from the time of the first symptoms till death it was not 3 months but closer to 9 or 12 months. He had, then, weakness, malaise and fatigability for several months before admission. It is stated in the past history that he had polyarthritis at the age of 33. I do not think that we can be sure that he had acute rheumatic fever, and there is no further history of other symptoms of acute rheumatic disease. One other thing not mentioned is that he had been treated in the Nerve Clinic on several occasions during the past 2 years.

I did not see this man when he first entered the hospital, but when I came on service he presented the picture of an acute, fulminating infection with classical symptoms of meningitis—stiff neck, high temperature, coma and an increased cell count in the spinal fluid. Several lumbar punctures were done subsequently. On physical examination there was one extremely important finding. The fundi were negative at the routine examination with the ophthalmoscope but a thorough search of the vessels and eyegrounds after the installation of homatropin showed one white patch on the left temporal margin. Such a patch is associated in my mind with either of two things, a tubercle or a leukemic infiltration. The first condition is a very rare one, and we know that only about 5 per cent of patients with tuberculous meningitis show tubercles in the fundi. The incidence of retinal infiltration in leukemia is considerably greater. The definitely circumscribed white patch, which was verified by several observers, persisted over a period of 10 days. During this time it changed from white to pale yellow. Just before this conference I mentioned this fact to Dr. Norcross, and he tells me that on the second admission a thorough search was made in the fundus for this patch and it was not found. We have then one very important observation, namely, the white circumscribed area in the fundus, which was observed and disappeared under observation.

The findings of the heart in relation to the polyarthritis at the age of 33 were extremely interesting. We did not believe that the signs in the chest were related to decompensation from a failing rheumatic heart. The patient was able to lie flat in bed at all times. In the chest the rales were heard posteriorly and were thought to be due to either leukemic infiltration

or miliary tuberculosis and not to decompensation from rheumatic heart disease.

There was considerable discussion between the service and the x-ray department as to the significance of the films of the chest. They were taken on a portable machine, and we interpreted things that the x-ray people did not confirm. On physical examination changes were observed in the chest that were not reflected on the x-ray plates until about 10 days later.

X-ray Interpretation

Dr. George W. Holmes: This is the sort of case on which an x-ray diagnosis can be made only with considerable study. This film I would interpret as being negative, except for slight changes, which might be enlarged blood vessels or lymphatics. I would not think it represented a miliary process in the lung or any other parenchymal lesion. I should say the heart was a little enlarged. This genitourinary set of films is certainly more abnormal, but they are portable films taken with the films behind the patient's back and there is quite a bit of motion. The thing that I cannot understand is why there should be such a difference in the film taken over a period of 5 days.

Dr. Talbott: The clinical signs changed very rapidly at that point. The two diseases mentioned on the x-ray requisition were miliary tuberculosis and leukemic infiltration of the lungs. The x-ray interpretation said that neither was present.

Dr. Holmes: Certainly if you compare these two films taken 5 days apart there is quite a definite increase in the process. The theory that the slight variation from the normal seen was due to blood vessel changes or infection that had occurred a long time ago would not explain the picture. It now appears to be a progressive process involving both apices or both upper lobes. It is much more marked in the upper lobes than the lower lobes. I should now think that tuberculosis would have to be very seriously considered.

Dr. Talbott: What would you say about the last plate?

Dr. Holmes: This film is negative, and I would now return to my original diagnosis of no active disease in the chest.

Differential Diagnosis Continued

Dr. Talbott: That does not help much. There are two or three things about the blood studies that I think we might mention. With a normal red count of 5,300,000 the hemoglobin is only 75, which means that the concentration of hemoglobin in the cells is probably below normal. The white cell count is 14,000 and the differential is definitely abnormal, with 32 per cent monocytes and 53 per cent polymorphonuclears. The Widal test was done because typhoid fever was considered as a possibility. The positive Widal did not disturb us. We felt that he did not have typhoid, although the association of high fever, enlarged spleen, weakness and malaise, with signs in the chest, were consistent with typhoid fever. The agglutination test for undulant fever was negative. The changes in the spinal fluid were interesting. The lumbar puncture was normal. At no time was there increased pressure. The increase in cells, however, was very striking. In the first fluid there were 4 cells, in the next 35 or 40 and up to 80, in the last 100. When the patient came back 6 weeks later, the spinal fluid was normal. The increase in cells, which developed under our eyes, was not observed at the subsequent admission. We searched the spinal fluid repeatedly for tubercle bacilli. We did not observe any pedicle and were not able to isolate the organism. The electrocardiogram showed auricular tachycardia. We will discuss this later.

The type of fever that this man showed was striking. One could separate the fever into two cycles—a short cycle, 1 or 2 days in duration, and two cycles of the Pel-Ebstein type. The clinical picture on admission, as I have said, was that of meningitis with definite meningeal involvement. The patient then began to get better, and he was taken off the danger list. Some time later, when we were making the visit on another ward, the head nurse came and said the patient was dying and asked us to come at once. We went immediately and found him very sick, extremely apprehensive, breathing rapidly, with pulse over 200 but perfectly regular. A diagnosis of paroxysmal tachycardia was made, and he was given ipecac. He vomited in characteristic fashion, and his tachycardia disappeared. He repeated this three or four times in the next 2 weeks. We found out later that many of the things he complained about during the periods of paroxysmal tachycardia on the ward were the things that made him go to the Nerve Clinic. The change in the red cell count from 5,500,000 to 3,500,000 during the stay in the hospital was a little unusual but not especially so. We see people with acute febrile conditions that have some damage done to the blood-forming organs or have inadequate ingestion of foods possibly related to the drop in red count during the period of fever. He apparently made a fine response to transfusion, because we see on a subsequent admission that the red count was 6,600,000. He had one attack of epistaxis and no other evidence of bleeding—no purpuric spots. The interesting thing is that he was apparently well from the time he left the hospital until a few days before his second admission. He was discharged without diagnosis, and I did not see him after that time.

On the second admission he was well devel-

oped and nourished. He did not look cachectic. He did not present the picture of a chronic illness but of an acute illness of a few days only. At this time pea-sized inguinal glands were noted. Thorough search at the first admission revealed no evidence of glandular adenopathy, and I think this represents a definite change. Increase in the size of the liver is again evidence that something was happening to his reticuloendothelial system. There was no change, however, in the size of the spleen. He was febrile on the second admission, had a pulse deficit, and again had rales at the bases. The red count was 6,600,000. I would like to know whether that is correct.

DR. FRANCIS T. HUNTER: Yes. It was 6,000,000 to 6,800,000, and down to 6,600,000.

DR. TALBOTT: My first explanation would be dehydration. When I saw the white cell count of 40,000 with the red cell count of 6,600,000, I thought of polycythemia, because we do see at certain stages of this disease a count quite consistent with this red count and a white count of 30,000, 40,000 or 50,000. I do not think, however, that he had polycythemia vera.

The smear at the second admission was interesting, and Dr. Mallory was kind enough to let me look at the smear yesterday. There are two or three points that I might mention. I did a rough differential and I counted over 60 per cent monocytes, monoblasts and blasts, whereas the count given in the abstract is only 42 per cent. The monocytes and monoblasts were fairly typical. Some of the monocytes were vacuolated, and the blasts all had pale cytoplasm. There were certain things in the red cells not mentioned. One was the presence of stippled cells. I found at least 1 stippled cell per high power field. There was considerable change in size and shape of the red cells with a few tailed forms. In other words, it was not the smear of polycythemia vera.

No mention is made of a sternal biopsy. I do not know whether it was done or not. The total protein of the spinal fluid was normal. There were no cells in the spinal fluid at the last examination. The patient again developed signs of meningitis and died.

I think that the differential diagnosis as we saw him on the ward and the clinical picture on the final admission lay between tuberculous meningitis and a form of leukemia. I do not think he had tuberculous meningitis. It seems unlikely that with all the studies which were done we would not have been able to find things consistent with this diagnosis. The spinal fluid sugar was normal at all examinations, and on the last examination the cells were normal and there was no increase in the protein. I have spent a little time in the library during the past 24 hours going over the literature and I find that leukemia with increase in the monocytic series is consistent with this diagnosis. The incidence of central nervous system findings in a monocytic leukemia is about 15 or 20 per cent. There are about 75 cases of monocytic leukemia reported in the literature.

I believe the diagnosis is acute monocytic leukemia with involvement of the central nervous system, liver, spleen and kidneys, and I think that he possibly had leukemic infiltration of his lungs, which would be consistent with the clinical findings. I would also like to make a diagnosis of rheumatic heart disease with mitral stenosis.

DR. RICHARDSON: This patient was on my service, and when I went off on vacation I was certain he had miliary tuberculosis. I was quite certain that he did not have mitral stenosis.

DR. BERNARD M. JACOBSON: I saw this patient about a week before he died. On the second admission I was asked to go up and see a case of bacterial endocarditis. After I had read through the history and physical findings and had made a sketchy physical examination of my own, I thought that the patient probably did have endocarditis. I looked at the second smear, the one with a high proportion of monocytes, and I saw these apparently immature cells but was not at all certain they represented the cells of monocytic leukemia or of any kind of leukemia.

I think the point Dr. Talbott brought out, that, in spite of the high red count, the red cells showed change in size and shape, is unusual and I do not believe I have seen it before. There was one finding in the smear that no one else mentioned, either on the first or second admission. It caught my eye rather accidentally. There was a moderate degree of rouleaux formation of the red cells and repeated smears showed rouleaux formation, at first thought to be due to dirt on the coverslip. We made a fresh moist preparation, and examination in the counting chamber showed that the rouleaux were still present. The only condition in which I have ever seen so much rouleaux formation is multiple myeloma, regardless of the level of the red count. Rouleaux formation of red cells has been reported previously in multiple myeloma. However, the conditions were very much against multiple myeloma in this case. The only disease I could think of that was like myeloma in its infiltrating properties in the bone marrow was leukemia. I thought it was an aleukemic leukemia of some sort.

DR. WALTER BAUER: I saw this man on one occasion on teaching rounds and it was obvious that he was critically ill on the first admission. He had pulmonary signs in addition to signs and findings suggesting meningeal irritation. Then he became afebrile and made this perfectly remarkable recovery with 15 or 20 pounds gain in weight and a return to a normal red count. He then returned to the hospital with a

picture not unlike that at the first entry. I wondered, no diagnosis being certain, whether we might possibly be dealing with a man that had had a right-sided endocarditis and whether the rapid changes in the pulmonary findings might be explainable on an embolic basis. I thought that even the monocytes might be part and parcel of the endocarditis, although I do not remember having seen a case of subacute bacterial endocarditis running a mononucleosis of as high percentage as in this case. Not feeling at all too certain I made the suggestion of right-sided endocarditis as a possible diagnosis.

DR. HUNTER: The day before I was presented this man's blood for examination I was talking to the men on the service about endothelial phagocytes and spoke of the fact that I had seen them in the peripheral blood in only two conditions, leukemia and bacterial endocarditis. I looked at the smear and decided it was that of monocytic leukemia. Dr. Bauer and I had a few words, and I still held out that he had monocytic leukemia although he could have bacterial endocarditis, too.

DR. TRACY B. MALLORY: Dr. Kubik, you saw him in reference to the question of meningitis.

DR. CHARLES S. KUBIK: I remember very little about the case. However, on going over the record now it is noted that headache had been present for a long time when the first lumbar puncture was made and showed a normal spinal fluid. Later on, the cells found in the spinal fluid would have fitted in with tuberculous meningitis but normal values for chlorides and sugar did not. Leukemic involvement usually affects the meninges but may affect both brain and meninges and so could explain headache with, at the start, a normal spinal fluid and later fluid-containing cells.

DR. MALLORY: There is one other type of examination that was done on this case—supravital studies. Dr. Gall, will you tell us about them?

DR. EDWARD A. GALL: I studied his blood on two occasions at the first and second admissions. On neither occasion did I find rouleaux formation, such as Dr. Jacobson has described. It is not infrequent that one does find rouleaux immediately after the preparation has been placed under the cover slip. I usually wait 10 or 15 minutes to allow the blood to settle, and perhaps that is why I did not observe them in this case.

At the first admission the blood contained normal monocytes and with them cells that have been recognized as epithelioid cells. There were no monoblasts at that time. These epithelioid cells have been noted previously in the blood, and Blackfan and Diamond have stated that they are pathognomonic of tuberculosis in children. They have been observed, however, in many other conditions, and I do not feel so confident about using them as a diagnostic measure.

On the second admission a definite shift to the left was noted, if such an expression can be applied to the monocytic series. In addition to monoblasts, which were present in significant numbers, there were also monocytes and epithelioid cells, which were evidently end stages in the process of differentiation. In addition there were phagocytic histiocytes and also certain tumor cells which could not be identified. Phagocytosis in the histiocytes was limited to detritus, and there was no phagocytosis of intact red cells. On the other hand, there was also present a high red count, which was a point against the diagnosis of leukemia. The polymorphonuclear cells, which would to a certain extent indicate the presence or absence of infection, showed no shift. A differential count on a dried smear showed a very small percentage of band forms, which was considered a very definite argument against the presence of infection. Therefore, on the basis of the monocytes, which were differentiating all the way through from monoblasts to perfectly mature forms and atypical mature forms, a diagnosis of monocytic leukemia was considered established.

DR. SCOTT: Did this patient show any bone change in the x-ray to rule in or out any diffuse form of Hodgkin's disease?

DR. MALLORY: I do not think so.

DR. TALBOTT: He did not have any bone changes at the first admission.

DR. SCOTT: Could not Hodgkin's disease without very much external lymphadenopathy give a picture something like this?

DR. MALLORY: We have recently had a case that we thought was Hodgkin's disease with a blood that came as near this as anything I have ever seen. I think that is a good suggestion.

CLINICAL DIAGNOSES

Rheumatic heart disease with mitral stenosis.
Subacute bacterial endocarditis?
Subacute leukemia, possibly monocytic?
Abdominal lymphoma?

DR. JOHN TALBOTT'S DIAGNOSES

Monocytic leukemia with central nervous system involvement.
Rheumatic heart disease, mitral stenosis.

ANATOMIC DIAGNOSES

Monocytic leukemia.
Atypical Hodgkin's disease?
Rheumatic heart disease, mitral and aortic, with mitral stenosis.
Acute endocarditis, terminal.
Bronchopneumonia.
Arteriosclerosis.
Prostatic hyperplasia, focal.

Pathological Discussion

Dr. Mallory: The autopsy on this man showed a very large spleen, weighing 700 grams, a very slightly enlarged liver, weighing 1,700 grams, and a slight generalized lymph node enlargement. The heart showed a well-marked mitral stenosis and an old fusion of two of the aortic cusps, which we thought probably was rheumatic. On the mitral valve there was an acute vegetation, which was undoubtedly bacterial. There was a terminal bronchopneumonia.

The final diagnosis has to rest on the microscopic examination, which proved very interesting. The most striking findings were in the spleen. It was packed with abnormal, atypical cells, many of which, however, could be definitely recognized as belonging to the monocyte series, and there seemed to be transitions between them and the blast forms and also large multinucleated cells, which would make one think of Hodgkin's disease. The bone marrow showed a significant degree of involvement, although it was not diffusely replaced with leukemic cells. The peripheral lymph nodes showed a great many mature monocytes but none of the early blast forms.

The two diagnoses that must be considered are monocytic leukemia and an atypical Hodgkin's disease with a terminal monocytosis. As I said, we have recently had a case of Hodgkin's in which in the terminal stages large numbers of monocyte-like cells appeared in the peripheral blood. However, in the reported cases of monocytic leukemia—for instance, cases 1 and 6 in the paper of Doan and Wiseman*—the resemblance of the histologic picture in the spleen and lymph nodes to that of Hodgkin's disease has been emphasized. It would be idle to speculate, at the moment, as to whether the two diseases are genuinely related. I think we may fairly say, however, that this case falls within the bounds at present assigned to monocytic leukemia by competent hematologists, and I am making that my final diagnosis.

Dr. Hunter: Are you calling this bacterial endocarditis, too?

Dr. Mallory: Yes, though I think that was only terminal.

Dr. Hunter: That is interesting because this is the fourth case of leukemia with bacterial endocarditis we have had. Another interesting point is the increase in red count, the apparent polycythemia. We have three cases with myelocytic leukemia and polycythemia. I do not know whether the combination has ever been noted before in monocytic leukemia.

A Physician: Did the brain show anything?

Dr. Mallory: It was negative grossly. There was no leukemic infiltration in the meninges. The lungs were negative except for terminal bronchopneumonia.

*Doan, C. A., and Wiseman, B. K.: Monocyte, monocytosis, and monocytic leukosis. Ann. Int. Med. 8:383 (Oct.) 1934.

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MATERNAL MORTALITY IN BOSTON

FOR some time the high maternal mortality in this country has been attracting attention, but it has been very difficult to ascertain the facts. Some isolated points can be determined easily, but there has been a notable absence of pertinent and adequate information. It has been alleged, as a result of comparative studies, that the maternal mortality is appallingly high in the United States. But is there sufficient uniformity of any sort, even in classifying diseases, to make comparative studies of any value? How can one compare the United States with Holland, for example?

In recent years studies have been undertaken in several cities in the United States, which have had as their object not so much comparison with other cities, as intensive investigation of morbidity and mortality. Of these studies the latest, and in some respects the most notable, is that which is now reported from Boston for the years 1933, 1934 and 1935. The amount of work involved in making this study can be appreciated only by one that has undertaken similar investigations, and what has been accomplished is due to patient, persistent effort, to a fine spirit of cooperation and to a willingness to face facts. Complete cooperation by all physicians whose patients died would be Utopian, but it is to the credit of the medical profession that cooperation was given freely in so many cases.

What lessons are to be learned from this study? First, the medical profession in Boston is practicing a great deal of bad obstetrics! How much is a great deal? More than New York or Philadelphia or Chicago or London or Paris or Berlin? Any bad obstetrics in Boston is a great deal, no matter what is done in other cities.

The fault lies first with the practicing physicians. Perhaps they generally do the best they know how, but it is not ignorance alone, it is often poor judgment, that brings the catastrophe. The fault lies next with the medical schools that fail to give adequate training to the students. Perhaps they are doing the best they know how, but for years there have been voices crying in the wilderness proclaiming the coming judgment. Obstetric education has been weighed in the balance and found wanting. However, the difficulty sometimes experienced in securing sufficient clinical material to provide adequate experience must not be overlooked.

Then the fault lies with the public, which in Massachusetts for so long a time refused to insist that a physician shall have received his medical education in an institution reasonably well equipped to give it, and, in fact, insisted that adequate standards of medical education should *not* be established for the Commonwealth. And again the fault lies with the public in refusing to recognize that much of the morbidity and mortality connected with childbirth can be prevented if the pregnant woman places herself under the care of a competent physician early in pregnancy.

The study will be perhaps of greatest value to the medical profession in indicating to them some of the points at which this exceedingly complex problem is to be attacked.

AUTOMOBILE ACCIDENTS

THE lowering of the number of automobile accidents, involving the death of tens of thousands and the injury of hundreds of thousands annually in the United States, is a problem requiring immediate and drastic action.

Of the three factors that are primarily responsible — the automobile, the pedestrian and the operator—the automobile is rarely at fault. Illegal lights and defective brakes at times re-

sult in accidents, but such conditions are really due to the carelessness of the operator or owner. Furthermore, the semiannual inspections required by many states have resulted in a decrease in the number of improperly equipped or adjusted automobiles.

A large percentage of the accidents in which pedestrians are killed or injured are absolutely unavoidable, so far as the operator is concerned. This disregard of the dangers of traffic and of the rights of the operator by some adults is difficult to understand but, in many instances, is undoubtedly due to one or more of the same individual characteristics possessed by faulty operators. It is difficult to conceive of any step that would prevent accidents of this sort. The irresponsibility of the normal child is comprehendible, and it is gratifying to learn that educational measures in the primary schools of Massachusetts have resulted in an appreciable decrease in the number of accidents involving child pedestrians.

In the majority of instances, however, the operator alone is to blame — "an operator" would be, perhaps, more accurate for frequently death or injury concerns the occupants of a properly operated automobile. At times the accident is due to flagrant disregard of state, city or town ordinances, at times to carelessness or inattentiveness, at times to recognized or unrecognized physical or mental defects and at times to pure selfishness and disregard of the rights of others, be they pedestrians or operators. How can this menace — the faulty driver—be removed from our highways?

Irrevocable cancellation of individual licenses would seem to be the only measure with sufficient threat to accomplish the desired effect. The lawbreaker or the operator involved in an accident because of carelessness or inattentiveness or because of selfishness or disregard of others might be penalized a certain number of points, depending on the seriousness of the conviction or of the accident. When the sum of demerits reached a predetermined total, cancellation would automatically take place. Credits might be allowed for years without conviction or accident. In fairness to all, "fixing" would have to be abolished, and some impartial board of appeal would have to be created.

Furthermore, every operator involved and surviving an accident and every operator above a certain total of demerits should be required to pass a rigid physical and mental examination before their licenses are reissued. This should include carefully conducted neurologic, psychologic and ophthalmologic tests, and with the latter the possibility of "night blindness" as suggested by Jeghers in this issue of the *Journal*, should not be overlooked.

The speedster, the driver that always tries to "beat the lights", one that passes on curves and hills, the "road hog", the inebriate or anyone involved through his own fault in a serious accident is a menace on our highways, and, after fair warning, should be outlawed. This demands unpopular legislation, but the public should place protection ahead of unwarranted personal privilege.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

JEGHERS, HAROLD. M.D. Western Reserve University School of Medicine, 1932. Instructor in Medicine, Boston University School of Medicine. Address: 80 East Concord Street, Boston University School of Medicine, Boston, Mass. His subject is "Night Blindness Due to Vitamin A Deficiency: A Consideration of Its Importance in Traffic Problems." Page 51.

LOMAN, JULIUS. M.D. Tufts College Medical School, 1925. Instructor in Neurology, Tufts College Medical School. Research Associate in Psychiatry, Boston State Hospital. Junior Visiting Neurologist, Beth Israel Hospital. Address: 353 Commonwealth Avenue, Boston, Mass. His subject is "Progress in Neurology in 1935." Page 56.

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CASE HISTORIES

NO. 2. INEVITABLE AND INCOMPLETE MISCARRIAGE

Mrs. M. G., a 37 year old Jewish housewife, reported for prenatal care in her fourth pregnancy on August 12, 1936. Her last period had begun on June 15, 1936, since which she had had no vaginal discharge until a few days previously, when she had noted a slight dark-brown staining. There had been no nausea or vomiting, breast symptoms or abdominal pain. Urination had been slightly more frequent than usual, and her bowels had moved daily without laxatives.

Her past history indicated that she had always enjoyed good health and that she had

*A series of selected case histories by members of the Section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

never suffered from scarlet fever, rheumatic fever or pneumonia. She had had no operations. Menstruation had begun at 14 and was of the 28 day type; each period was moderate in amount, painless and lasted 4 days. She had had three previous pregnancies; the first terminated some two weeks prematurely with the normal birth of a 5 lb. 8 oz. child and the second and third had gone to full term, the last resulting in the birth of an 8 lb. 12 oz. infant. None of the pregnancies had been complicated by toxemia or bleeding, and all the children were alive and well. She had never had an abortion or a miscarriage. The patient's family history gave no evidence of tuberculosis, diabetes, cancer or insanity.

At her initial visit the patient weighed 135 lbs. Her blood pressure was 122/70, and her urine was free of albumin and sugar. Further physical examination was not made at this time, because the recent vaginal discharge implied a threatened miscarriage, which might be made inevitable should any pelvic manipulations be carried out. Accordingly, the patient was directed to remain in bed at home. Lavatory privileges were denied, visitors were excluded, and all factors that might cause pelvic congestion, either directly, such as coitus, or indirectly, such as enemata or catharsis, were warned against. A low residue diet was outlined.

For the next 5 days the patient's pulse and temperature were normal. She slept well, and her bowels moved daily. There was an intermittent, slight vaginal discharge, somewhat brownish in character, but no cramps occurred. Gentle abdominal palpation during this time showed no tenderness, masses or spasm.

On the evening of the sixth day the husband telephoned to report brisk vaginal bleeding, with clots. The patient was transferred at once to the hospital for further examination and treatment. There, under aseptic precautions and without anesthesia, vaginal examination revealed a moderate flow of bright red blood, mingled with clots. The cervix was soft, the os admitting one finger, while through it was felt protruding a small piece of tissue. The uterus was enlarged to the size of a pregnancy of 2½ months' duration and was anterior, soft, freely movable, slightly irregular and nontender. The vaults showed no masses or tenderness. The examining finger, when withdrawn, brought away several fragments of what was believed to be placental tissue. Since the entire picture indicated an inevitable and incomplete miscarriage, curettage was decided upon to insure evacuation of the uterus and to control the continued bleeding.

Under gas-oxygen anesthesia, the vagina and perineum were prepared with ether followed by 5 per cent alcoholic picric acid. A weighted speculum was placed in the vagina, and the cervix was grasped with a double-hooked tenaculum and drawn into view. Some large pieces of placental tissue were removed from the uterine cavity with a long clamp. The cervix was stretched with a Goodell dilator sufficiently to admit a large, dull curette; with this instrument the entire interior of the uterine cavity, anteriorly, posteriorly, laterally and across the fundus, was thoroughly curetted. Since the patient had a normal pulse and temperature, a sharp curette was also used for this purpose, scraping the uterine walls until they felt hard and until the characteristic faint rasping sound of firm uterine tissue was obtained. The uterine cavity was then wiped out, first, with a strip of plain gauze, and, finally, with a strip of gauze soaked in tincture of iodine. An ampoule of pituitrin was administered intramuscularly at this time, followed ½ hour later by an ampoule of intramuscular ergot. All instruments were removed, and the patient returned to bed in good condition with her pulse 100. One sixth grain of morphia was given to control postoperative restlessness and discomfort.

During convalescence the patient was allowed house diet on the second day. Fluid extract of ergot was given in 1 dram doses three times daily for the first 3 days. Enemata and catharsis were prescribed after the third day. Her chart was uneventful, save that on the fourth day the temperature rose to 101°F. with the pulse 90 and respirations 20. No chills occurred. Physical examination showed no abnormalities; the abdomen was not tender. The white blood cell count and urine were normal. The vaginal discharge was subsiding and normal in character. No cause was found to account for this episode save that the bowels had not moved and that the patient had had an emotional upset at the time. The temperature rise receded within 48 hours, and the patient was allowed out of bed three days later. She was discharged home on the tenth day.

The pathologist reported the curettings to consist of decidua vera, chorion and villi from a pathologic ovum.

Discussion. The terms abortion and miscarriage are loosely used in ordinary parlance to indicate interruption of pregnancy before the end of the twenty-eighth week of gestation, when the so-called period of fetal viability begins. Strictly speaking abortion occurs before the complete formation of the placenta at the sixteenth week, and miscarriage from the sixteenth to the twenty-eighth; this case, therefore, should properly be termed an abortion.

The physician acted properly in not examining the patient's pelvis at her first visit. At the time, though the evidence available from the

history pointed to the fact that the patient was pregnant, the brown vaginal discharge indicated, presumably, a beginning separation of the ovum from its implantation; furthermore, the lack of abdominal symptoms and the lack of tenderness in the abdomen, as later demonstrated, yielded evidence that the pregnancy was probably not ectopic. The initial policy, therefore, of treating the condition as a threatened abortion was entirely justified. As to the immediate diagnosis of a degenerated or pathologic ovum, laboratory resources are not at present of reliable clinical value, save in the rare cases where hydatidiform mole is suspected.

After a course of conservative treatment lasting six days, the patient began to bleed "briskly". No cramps are mentioned in this connection, but cramps often do not occur unless the ovum is completely or nearly completely separated and free in the uterine cavity. Since the hemorrhage was marked, the obvious treatment was carried out, that is, to examine the patient, confirm the diagnosis, which by then had changed from threatened to, presumably, inevitable abortion, and to evacuate the uterus, in order to allow the emptied organ to contract and control the bleeding. The examination revealed the os patulous to one finger, this fact indicating that the abortion was inevitable; it also revealed placental fragments coming through the os, this indicating that the abortion was incomplete.

Curettage was therefore carried out under surgical anesthesia; it may be assumed that the condition of the patient as a risk for anesthesia had been ascertained beforehand. That portions of the placenta were lying loose in the uterus may be inferred from the fact that they were removed by the clamp. The balance of the placenta was separated and removed by the curettes. The use of the sharp curette was of questionable judgment, both from the standpoint of the danger of perforating the uterus and from the fact that the uterine contents were probably not free of bacteria, despite the normal temperature and pulse of the patient. In the latter instance, a sharp instrument may act as an efficient propagator of infection into the uterine wall.

Following the curettage the bleeding was immediately controlled by uterine contraction and retraction, aided by the oxytocic medication employed. The convalescence of the patient was not remarkable, save for the febrile episode on the fourth day; the cause of this is rather speculative.

The diagnosis of a pathologic ovum was implied at the time of curettage, when evidently no fetus was recognized, and confirmed finally by examination of the curettings.

MISCELLANY

DR. STRONG HONORED

Dr. Richard P. Strong, Professor of Tropical Medicine, Harvard Medical School, has been elected an Honorary Fellow of the Royal Society of Tropical Medicine and Hygiene, and has been invited to give the first Chadwick Lecture in London in January, 1937.

DR. NORTON APPOINTED SUPERINTENDENT OF THE BOSTON STATE HOSPITAL

Dr. Harold F. Norton, of Hyde Park, senior physician at the Boston State Hospital, has been named as superintendent of the institution to succeed Dr. James V. May, who has recently retired on pension.

Dr. Norton, who is 36 years old, is a graduate of Boston College, Tufts Dental School and Harvard Medical School. Since his graduation he has been associated with various hospitals for mental diseases in Connecticut and in Massachusetts.

DR. OVERHOLSER APPOINTED RESEARCH DIRECTOR

On January 3 the National Committee for Mental Hygiene announced the appointment of Dr. Winfred Overholser as director of the committee's division of mental hospital research.

His duties are to include a nationwide survey of research problems now under way in such institutions and the discovery and listing of leads worthy of development. He will be assisted by an advisory committee including Dr. C. O. Cheney, of New York; Dr. Adolf Meyer, of Baltimore; Dr. Douglas Singer, of Chicago, and Dr. C. F. Williams, of the South Carolina State Hospital.

It is gratifying to learn that Dr. Overholser's training and ability have been appreciated and properly recognized.

TUBERCULOSIS ABSTRACTS

JANUARY, 1937—No. 1

Medical treatment alone is not always enough to insure the recovered tuberculosis patient against relapse. When he returns from the sanatorium to the care of his own physician he is faced with the difficult problem of adjusting himself to normal living and to regular employment. How well he succeeds in making this adjustment will depend largely on what has happened to him during his period of curing. The newer emphasis on social diagnosis and treatment which is being introduced in many sanatoria will therefore be of interest to the general practitioner who has the difficult job of keeping his patient well.

TREAT THE WHOLE MAN

The prevention of relapse among arrested cases of tuberculosis is a chief concern of those engaged in tuberculosis work. Various schemes such as shel-

tered workshops, convalescent homes and camps have been set up from time to time but they reach only about one out of every seventy-five patients who need such service. It is essential that some practical program be worked out that can be applied on a much wider scale.

In an effort to escape from theory and opinion and to build a program based on the actual needs of patients, a factual study was made of 5,000 patients over 16 years of age in 40 sanatoria in 15 states. The results show that the majority of sanatorium patients are young; with limited schooling; with occupational experience largely in the semiskilled or unskilled group; in need of vocational guidance; with definite desires for further study and training. In the light of these findings it was decided to stop using the term "after-care" since by waiting until after the patient has been discharged from the sanatorium, opportunity for guidance and training is lessened and sometimes lost. "Social and vocational rehabilitation" more accurately describes the ultimate goal, which is "to return each patient to that place in society where he can obtain the greatest amount of happiness for himself, at the same time that he is giving his best to the common life around him."

Guidance involves a knowledge of the individual to be guided and a knowledge of the community in which he is to live. Proficiency in a certain type of work and enjoyment of that work usually go together. It is important to find each patient's strong points and to give him all the training he can take in line with his best abilities. Vocational adjustment, important for everyone, may be a deciding factor between illness and health in the case of the tuberculosis patient.

While aptitude tests are of great help in counseling much can be accomplished without them. Careful study of school life, in terms of subjects liked and disliked; of occupational experience in terms of satisfaction or dissatisfaction; of leisure including favorite sports and amusements, preferred magazines and newspapers, will throw a great deal of light on the patient's abilities. It is equally important to get at the patient's tastes, and his hopes and ambitions for the future. Knowledge of work opportunities in the community is of course essential to the giving of sound vocational advice.

The methods outlined above are called "social and vocational diagnosis". Just as medical treatment is undertaken only after a complete medical diagnosis has been made, so social plans should be made only on the basis of a diagnosis of his social and vocational needs.

Complete physical rest cannot be obtained without peace of mind. Planning for the future often allays most of the patient's persistent worries, thus facilitating the medical cure. Planned study is proving its therapeutic value in many sanatoria. Teachers are furnished by the sanatorium, through WPA funds, depending on the resources of the community. Instruction is brought to the bedside by tutors and through radio. Some sanatoria have their own broadcasting system so that patients in the different wards throughout the hospital can be included in classes. Regular grade school work, high school and college subjects and a number of vocational courses are being taught.

After discharge the study begun at the sanatorium is carried on through outside agencies, notably through the State Rehabilitation Bureaus which exist in 44 states. These bureaus have public Federal and state funds for the retraining of people who because of accidents or illness should not return to their old occupations.

But the best guidance and training is barren unless it results in actual placement. The widening Public Employment Service is deeply interested in fitting people who have recovered from tuberculosis into suitable employment. The recovered patient is not treated as a handicapped individual. His assets not his liabilities are stressed. In days of vast unemployment it is more than ever essential that the recovered patient be trained along the line of his greatest capacity.

Patients are often told to return to their old occupations, but for a very large number of them this is not possible. A recent study of more than 300 patients revealed that two-thirds of them must be trained for new occupations. There is no simple formula for this solving of the problem of rehabilitating the tuberculous. The problem is social as well as medical. Medical diagnosis and social diagnosis, treatment and training, recovery and placement in a suitable occupation are essential elements in the solution of the problem of rehabilitation and the prevention of relapse.

Burhoe, Beulah W.: Rehabilitation of Sanatorium Patients in the U. S. A. Bull. de l'Union Internationale Contre La Tuberculose 13: No. 1 (Jan.) 1936.

What Is Going on Now in Rehabilitation

During the last year and a half there has been a marked increase in interest in rehabilitation throughout the country. There are several full-time programs which not only are accomplishing valuable results but which are blazing new trails in this field.

In Cincinnati the rehabilitation program is under the direction of a full-time Director of Social and Economic Services, whose salary is paid by the county commissioners. A fine educational building has been erected on the grounds of the sanatorium. The program includes vocational guidance and counseling, academic and prevocational training, and occupational therapy. There is a close tie-up with the various social agencies and with the local employment service so that valuable methods of solving difficult problems are being worked out.

In the District of Columbia the rehabilitation program is one in which there is close cooperation with the community agencies which have a stake in the rehabilitation problem. The work is under the management of a director, whose salary is paid out of Christmas Seal Sale funds. Testing and

counseling are given suitable patients, and adult education courses are being carried on in the sanatorium for both white and colored patients under the emergency education program.

In Saranac Lake a grant from the Carnegie Foundation made possible the setting-up of the Saranac Lake Study and Craft Guild. The Guild is conducting an ambitious program in adult education. A wide variety of class instruction is being offered and the majority of the teachers have been found among the patient group. A full-time psychologist is giving aptitude tests to determine the fitness for diversional and vocational courses.

A program of aptitude testing and counseling has been inaugurated in Fort Worth, Texas.

Last fall an important precedent was set in Indiana where for the first time social security funds were used to finance a program for the testing, counseling, education, and placement of tuberculosis patients.

A full-time program, financed by Seal Sale funds, will be initiated in Niagara County, New York. This project includes aptitude testing and counseling, the organization of adult education in the sanatorium and cooperation with community agencies aimed at the reemployment of discharged cases.

At the Mountain Sanatorium in Hamilton, Ontario, a counseling and testing procedure has been initiated in order to make the adult education program more effective.

If the rate of progress shown during the last year and a half can be maintained, it is not too much to hope that within a few years rehabilitation will take its deserved place in the program of the tuberculosis associations throughout the country.

AN ERROR IN DIAGNOSIS

Twenty years ago a man named Balthazar Balmint went to a hospital in London for treatment. It was discovered that he was suffering from a very rare bone disease, which would probably end his life very soon. The doctors offered to pay the sick man, on condition that he would will his body to them, one pound sterling per month during his life, and to give his heirs seven pounds sterling. However, Balmint is still living; the hospital continues to pay him an allowance, and this false diagnosis has already cost about 18,000 francs.—*Naturisme* (Paris), quoted in *New York State J. Med.*

BIRTH RATE DECLINING IN UNITED STATES

The birth rate in the United States continues downward, latest figures analyzed by statisticians of the Metropolitan Life Insurance Company show. The 1935 birth rate of 16.8 births per thousand population almost reached the all time low of 16.6 recorded in 1933.

The slight rise in 1934 encouraged the hope that the long downward trend and the threat of an ultimately declining population might be averted. The latest figures show, the statisticians point out, that the 1934 rise was only a temporary deviation from the general downward trend of the birth rate.

Each of 33 states and the District of Columbia, representing over half the country's population, showed a falling birth rate. Nine states which showed increases in 1935 were the following: Illinois, Michigan, Wisconsin, Arizona, Colorado, Montana, California, Florida and Mississippi. These states make up 21 per cent of the population of the country.

Canada's birth rate is declining even more markedly but this nation can better afford the decline as she has a somewhat higher birth rate than the United States. The Canadian birth rate for 1935 was 20.2 per thousand.—*Science News Letter*, October 17, 1936.

NEW RABIES VACCINE IS SAFER MEANS OF PROTECTION

A new and safer weapon against rabies has been developed by Drs. Leslie T. Webster and Anna D. Clow of the Rockefeller Institute for Medical Research, it appears from their report to the journal, *Science* (November 27).

The new weapon is a rabies vaccine made from rabies virus cultivated in the laboratory. It protects mice against rabies "street" virus and promises to give the same protection to dogs from the rabies virus which they may pick up from other rabies-infected animals on the streets or elsewhere.

Rabies vaccines now in use are composed largely of animal brain or spinal cord tissue containing virus in either virulent or inactive form. The presence of nervous tissue in the vaccine, the Rockefeller scientists point out in their report, is not only unnecessary but potentially dangerous. It may produce paralysis or other unfortunate results following vaccination. The new virus does not contain nervous tissue.—*Science News Letter*, December 12, 1936.

CORRESPONDENCE

POOR JOHNNY REB AGAIN

Editor, *New England Journal of Medicine*,

In a former letter to the *Journal* I quoted the remarks of a poor and dysenteric Confederate soldier, concerning the wonderful equipment and shining "nuniform" of a union cavalryman, and also the wonder of regular bowels, free from dysentery, was pathetically touched upon.

Along these same lines the following quotation, from Margaret Mitchell's "Gone with the Wind," may be of interest to your readers.

The Civil War had just ended, and thousands of Confederate soldiers were "on the walk" back to their desolated homes in Georgia.

THE QUOTATION

"Old and young, talkative and taciturn, rich planter and sallow Cracker, they all had two things in common, lice and dysentery. The Confederate sol-

dier was so accustomed to his verminous state he did not give it a thought and scratched unconcernedly even in the presence of ladies. As for dysentery —the 'bloody flux' as the ladies delicately called it —it seemed to have spared no one from private to general. Four years of half starvation, four years of rations which were coarse or green or half putrified, had done its work with them and every soldier who stopped at Tara was either just recovering or was actively suffering from it.

" 'Der ain a soun' set of bowels in de whole Confedrut ahmy,' observed Mammy darkly as she sweated over the fire, brewing a bitter concoction of blackberry roots which had been Ellen's sovereign remedy for such afflictions. 'It's mah notion dat 'twernt de Yankees whut beat our gempmum. 'Twuz dey own innards. Kain no gempmum fight wid his bowels tuhnin' ter water.' "

Yours truly,

WM. PEARCE COUES, M.D.

January 7, 1937.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Illinois,
January 5, 1937.

Editor, *New England Journal of Medicine*,

In addition to the articles enumerated in our letter of November 27 the following have been accepted:

Calco Chemical Co.
- Crystal Violet Medicinal—Calco
- Crystal Violet Jelly—Calco

Eli Lilly & Co.
- Ampoules Pentobarbital Sodium—Lilly, 0.5 Gm. (7½ grains)

Merck & Co., Inc.
- Gold and Sodium Thiosulfate—Merck
  - Ampuls Gold Sodium Thiosulfate — Merck, 0.01 Gm.
  - Ampuls Gold Sodium Thiosulfate — Merck, 0.025 Gm.
  - Ampuls Gold Sodium Thiosulfate — Merck, 0.05 Gm.
  - Ampuls Gold Sodium Thiosulfate — Merck, 0.10 Gm.
  - Ampuls Gold Sodium Thiosulfate — Merck, 0.20 Gm.
  - Ampuls Gold Sodium Thiosulfate — Merck, 0.25 Gm.
  - Ampuls Gold Sodium Thiosulfate — Merck, 0.30 Gm.
  - Ampuls Gold Sodium Thiosulfate — Merck, 0.50 Gm.
  - Ampuls Gold Sodium Thiosulfate — Merck, 1.0 Gm.
- Ichthyol—Merck

Riedel-de Haen, Inc.
- Pernoston Tablets, 3 grains

Sharp & Dohme, Inc.
- Diphtheria Toxoid-Alum Precipitated

Nonproprietary Articles
- Crystal Violet

Yours sincerely,

PAUL NICHOLAS LEECH, *Secretary*.

RECENT DEATHS

ORDWAY—CLARENCE EUGENE ORDWAY, M.D., of Winchester, Massachusetts, died at his home, 371 Main Street, January 5, 1937, in his fifty-seventh year.

For some years Dr. Ordway had been chief of the staff of the Winchester Hospital. A native of West Hampstead, N. H., he was graduated from Winchester High School, Yale University and the Harvard Medical School.

He was a member of the Massachusetts Medical Society, the American Medical Association, Zeta Psi Fraternity, the Aesculapian Club and the Winchester Country Club.

Surviving him are his widow, one son and two daughters.

RICHMOND—ERNEST DALTON RICHMOND, M.D., of Reading, Massachusetts, died at his home, 24 Longview Road, January 4, 1937, in his sixty-eighth year.

A native of Charlemont, Dr. Richmond was a graduate of Williams College and of the University of Vermont College of Medicine.

He was a member of the Massachusetts Medical Society, the American Medical Association, the American Legion, the University Club of Boston and the Masonic Order.

SCHUBMEHL—FRANK EDWARD SCHUBMEHL, M.D., of 109 Broad Street, Lynn, Massachusetts, died at the New England Deaconess Hospital on January 5, 1937, in his sixty-ninth year.

A graduate of the Boston University School of Medicine, Dr. Schubmehl lectured on industrial medicine at the Harvard Medical School, and for many years was infirmary physician at the West Lynn and Everett divisions of the General Electric Company.

He was a retired member of the Massachusetts Medical Society, and a member of the American Medical Association and the Lynn Medical Fraternity.

Surviving him is his widow, Mrs. Marie A. Schubmehl.

NORTON—GEORGE EDWARD NORTON, M.D., of 16 Linnaen Street, Cambridge, Massachusetts, died at his home, January 6, 1937.

A native of Lowell, Massachusetts, Dr. Norton was a graduate of the University of Vermont and of Tufts College Medical School. He had been a practicing physician in Cambridge for more than thirty years and was a Fellow of the Massachusetts Medical Society.

Surviving him are his widow, Mrs. Nellie Norton, and one son, George Edward Norton, Jr.

NOTICES

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

The next written examinations and review of case histories of Group B applicants by the American Board of Obstetrics and Gynecology will be held in the various cities in the United States and Canada on Saturday, March 6, 1937.

The next general examination for all candidates (Groups A and B) will be held in Atlantic City, N. J., on June 8 and 9, 1937.

Application blanks and booklets of information may be obtained from Dr. Paul Titus, Secretary, 1015 Highland Building, Pittsburgh (6), Pennsylvania. Applications for these examinations must be filed in the Secretary's office not later than sixty days prior to the scheduled date of examination.

COMMONWEALTH FUND POSTGRADUATE MEDICAL FELLOWSHIPS

The Commonwealth Fund of New York City is offering the following fellowships at the Harvard Medical School, Courses for Graduates, Boston, Massachusetts. These are available to members of the medical societies of Massachusetts, Maine, New Hampshire and Vermont.

Medicine, given at the Massachusetts General Hospital, Peter Bent Brigham Hospital or Boston City Hospital. A group of at least six must take the course at one time. Such a group may be made up from any one of the four states in which the fellowships are offered, namely, Massachusetts, Maine, New Hampshire or Vermont.

Pediatrics, given at the Children's Hospital. Not more than two may take the course at one time. Fellows live at the hospital.

Obstetrics, given at the Boston Lying-in Hospital. Not more than two may take the course at one time. Fellows live at the hospital.

Office Surgery, given at the Boston City Hospital; designed for physicians engaged in general practice; subjects studied are surgical problems met in the office; instruction in the outpatient department. A group of at least six must take the course at one time.

Fellowships are for one month. The stipend is $250.00 plus tuition and traveling expenses from place of residence to Boston and return.

Qualifications: Applicant must be a graduate of a grade "A" medical school; a member, in good standing, of the Massachusetts Medical Society, the Maine Medical Association, the New Hampshire Medical Society or the Vermont State Medical Society. He must have been in practice at least five years, should preferably be under forty-five years of age and must be a resident of a community of less than 10,000 population. Application blanks may be obtained from The Division of Public Health, The Commonwealth Fund, 41 East 57th Street, New York City, or from the secretary of any of the above-mentioned state medical societies.

MEDICAL CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 p. m. on Thursday, January 21, in the Amphitheater of the Peter Bent Brigham Hospital, Dr. O. H. Perry Pepper, Professor of Medicine, University of Pennsylvania, and Physician-in-Chief, pro tempore, Peter Bent Brigham Hospital, will give a medical clinic. To it are cordially invited practitioners and medical students.

REPORTS AND NOTICES OF MEETINGS

THE NORFOLK DISTRICT MEDICAL SOCIETY*

ABSTRACTS OF PAPERS AND DISCUSSION

INDICATION FOR SPINAL FUSION: Dr. Thomas F. Broderick.

By spinal fusion is meant the union of two or more segments to deprive the part of movement as separate entities. Should an operation be performed where the symptoms are constant but increasing attacks of pain, and x-rays show a mechanical reason for these attacks? The surgeon says an operation should be performed in appendicitis since the cause is known and the treatment is efficient. The same situation obtains here. We have studied this condition for fifteen years and several hundred fusion operations have been carried out in my service.

A normal spine is one in which the bones are free from abnormalities and in which the articular facets are regular in size and shape. In the normal spine the neural canal exhibits a long gentle curving line and the articular facets are not weight bearing when the body is upright. In the so-called "relaxed" type of back the weight bearing line is shifted due to soft tissue. Besides the erector spinae muscle (sacrospinalis) and the intervertebral disks, only dense ligaments support the spinal column.

Over 50 per cent of all spines present abnormalities, but, of these, less than one-half offer symptoms necessitating treatment. A weak back is one which has a potential source of strain. In weak backs a small injury or even a change of occupation may serve to precipitate symptoms of common strain. Most abnormalities occur at the junction of a movable with a nonmovable part; for example, the lumbosacral articulation. Under certain conditions the spinal ligaments are lengthened.

X-ray pictures were shown illustrating various spinal abnormalities and the results of successful fusion operations. By these pictures it is possible to

*At St. Elizabeth's Hospital, October 27, 1936.

analyze spaces in the spinal column and estimate its ability to function.

Conclusion: The function of the spinal column depends on its bony structure and on its ligaments. Sooner or later postural irregularities will produce chronic back strain.

RECENT ADVANCES IN THE TREATMENT OF PRURITUS VULVAE: Dr. Charles J. Kickham.

The term, pruritus vulvae, is applied to the condition in which there is an itching in some part of the external genitalia. It is very resistant to treatment. It may be caused by: 1. Vaginal discharge due to senile changes in the vulva, chronic cervicitis, cancer, or the parasite *Trichomonas vaginalis*. 2. Skin disease. 3. Parasitic infection. 4. Irritating urinary contaminants. 5. Desquamated epithelial cells. 6. Chronic pelvic inflammation. 7. Leukoplakia.

The incidence of the disease is greater during later life. As the predominant symptom is itching the patients try to alleviate the discomfort by scratching, which gives rise to excoriated areas. The itching may spread to involve the internal aspect of the thighs and the region of the rectum. The patient becomes nervous and irritable and there is a general loss of tone due to insomnia, and in sensitive patients due also to subjective humiliation.

1. Vaginal discharge — If sufficiently abundant, *Trichomonas vaginalis* may be ruled out by the drop method. If found to be present a germicide should be used. The patient should be told to return for further treatments because the condition is usually recurrent. In chronic cervicitis or cancer the accepted methods of cautery and surgery should be employed. If due to senile changes, endocrine therapy may often be employed successfully.

2. If leukorrhea is found as a result of chronic pelvic inflammation, medical or surgical aid is required.

3. When the cause is a skin disease the case may need the services of a dermatologist. A helpful factor in making this diagnosis is a previous history of the skin disease. If pediculi are found, cleanliness is advised.

4. Cases of pruritus are never treated without a urinary examination, for diabetes is a common cause.

5. An important diagnostic feature of the inspection is to determine the presence of leukoplakia, that is, the white areas among angry red sections. Since cancer may easily develop from this, radical measures should be taken—perhaps even excision.

Besides local treatment, build up the whole body economy. Impress on the patient the necessity of persistence, that the doctor may be given the opportunity of studying the various types of treatment.

Locally one observes an indurated skin, small foci of infection due to scratching, reddened labia and eroded epithelium.

The treatment is antipruritic ointments, general tonics for the nervous system, and sedatives for insomnia. If the disease occurs after the menopause, endocrine treatment should be tried. My own experience with endocrines has not been so satisfactory as some of those reported in the literature.

I have used alcohol injections with very favorable results, with or without local anesthesia. The injection is intradermal but not subcutaneous. If the patient's circulation is poor, be more conservative with the amount of alcohol used. If the case is early and the blood supply is good, a fairly extensive area may be injected. Aseptic conditions are used and 3 to 4 minims are injected at one site. When the treatment is successful, all excoriations are removed. In conjunction with these alcohol treatments, build up the body by means of hygiene.

A patient who did not respond to alcohol injections was effectively cured by x-ray therapy.

If these methods fail, a radical vulvectomy may be performed. All the skin and nerve endings are removed by slashing the skin, though caution must be observed in removing the skin in the rectal area.

Conclusion: For functional pruritus we recommend the following treatment: 1. Alcohol injections. 2. X-ray therapy. 3. Vulvectomy. Mild ointments may help early cases, but the usual ones are late cases and are best treated as above.

Dr. Parvey: In menopause we have decreased activity of the ovaries, and pruritus vulvae is probably a result of this.

Dr. Parvey recommended the use of large doses of ovarian hormone in cases of senile pruritus vulvae.

MEDICAL ASPECTS OF GALLBLADDER DISEASE: Dr. John J. Whoriskey.

The incidence of gallbladder disease is very great. In a recent instance of 66 autopsied cases in which this disease was found, only 6 came to the table with the correct diagnosis. At the Mayo Clinic correct diagnoses were made in only 5 per cent of the cases. The surgical idea of "once a gallbladder disease always a gallbladder disease" is not always true. Removal of the gallbladder does not necessarily remove the disease. By removing this organ we remove an end-result.

The three factors to be considered in the treatment of gallbladder disease are disturbances due to infection, biochemistry, and stasis.

1. Infection may enter through the venous portal system, through disturbances of the colon, through the cystic artery, or through the lymphatics (ascending from an inflamed appendix). The bacteria (*Staphylococcus aureus* and *Staphylococcus viridans*) may arise from the appendix, teeth, and tonsils, or from the colon. The use of bacterial vaccines to combat these infections is still in the experimental stage.

2. A disturbance in biochemistry is associated with this disease which gives rise to a precipitation of calcium and bile pigments. The cholesterol concentration in the blood is raised considerably. Since the cholesterol content of the blood and bile go hand

in hand, the cholesterol in the bile is increased. The cholesterol in the gallbladder is held in solution by alkaline cholates. The inflamed mucosa may absorb the cholates allowing the precipitation of cholesterol, with a possible physiologic storm in its train. Exogenous cholesterol present in egg yolk, cream, herring and other fatty fish may increase the cholesterol content of the body.

3. The study of stasis offers a therapeutic approach to the problem. Gallbladder disease is alleviated by any attempt to promote increased activity of the gallbladder. Food in the duodenum is the most active in this respect and is better than anything else. We must assist gastric digestion. I get best results with magnesium oxide. Coated tongue, flatulence, and a tendency to mushy stools are best treated by adding a small amount of hydrochloric acid to relieve stasis. Cholagogues are other means of stimulating the gallbladder. Magnesium sulfate can be used and also a mixture of oleic acid and bile salts (on the market).

Conclusions: Three conditions of this disease are worthy of study:

1. Infection. Eliminate the focus of infection.

2. Chemistry. Try to control the formation of cholesterol when it is not endogenous.

3. Stasis. This offers the medical man the best chance to get good results. Gallbladder disease is primarily a surgical affair but should first be treated by the family doctor who can often relieve it and should always try to do so.

Dr. Jankelson: A plea was made for the use of bile salts in the treatment of gallbladder disease, for bile salts are the most important cholagogues. They should be given an intestinal coating. The most efficient is sodium dihydrocholate.

Surgical Aspects of Gallbladder Disease: Dr. Joseph Stanton.

A large per cent of chronic gastrointestinal diseases are due to the gallbladder. The function of this organ is to concentrate and store bile. A more reasonable hypothesis is that it serves to equalize the pressure between the ducts. This explains why the common bile duct enlarges when the gallbladder is removed.

If repeated tests show a rising bile an operation is dangerous; with a falling bile the chances of a successful operation are good. An eminent surgeon has stated: "No one is infallible in the differential diagnosis of all gallbladder disease, so save a life by operating." I say it is safer to wait unless there is an extreme tenderness with a rising white count. The diagnosis of acute gallbladder disease is easy. Jaundice, when present, clinches the diagnosis. When stones are present the pain radiates to the back.

I have used spinal anesthesia in all my operations; it gives better exposure and relaxation. I believe in cauterizing the bile duct. The common bile duct should be examined with great care for stones or enlargements.

Conclusion: From 1,000 cases of my own experience I can say that patients properly prepared can be handled in gallbladder operations with negligible fatalities.

Radiologic Aspects of Yeast Infection of the Lungs: Dr. Thomas R. Healy.

Yeast infection is more common than was realized a year ago. It is never caused by commercial yeast; ordinary household yeast is not pathogenic. The fungus is widely distributed but since the disease is rare, man must possess a high immunity. The predisposing factors are little known; it may occur by the inhalation of dust containing the mold. It may occur at any age in either sex, but is generally found in the male. The growth of the yeast plant causes rupture of the alveoli with subsequent pneumothorax. Most patients recover spontaneously from the disease which lasts from 1 to 3 months; sometimes the clinical course ends fatally. Yeast infection may be primary and not always secondary as some claim.

The diagnosis of yeast infection is not by x-ray alone. It is to be suspected when the x-ray shows severe lesions of the lungs without severe symptoms. The diagnosis is a clinical laboratory responsibility. The recovery of yeast bodies from a single sputum is not diagnostic. The yeast cells must be found in repeated tests.

X-rays were shown illustrating the changes in the lungs of patients with this affliction.

The Treatment of Pneumonia: Dr. John F. Casey.

Pneumonia is a disease with a natural course of recovery. Half the pneumonia deaths are due to the lobar type. Thirty-two types of pneumococci have been isolated but antiserums for only types I and II are useful. However, these two types account for many cases. Antiserums for some of the other types have been prepared but their action is not certain. Most cases of lobar pneumonia run a quick course. We now have a rapid means of typing by the Neufeld capsule technic. Treatment for types I and II: Get sputum to the laboratory immediately. Determine sensitivity of the patient to asthma or hay fever to determine his sensitivity to horse serum. (Not all asthmatics or hay fever patients are sensitive to horse serum.) Do not give serum to those who have had serum within 7 days. Felton was successful in concentrating serums so that we can now give a high concentration of antibodies in a small dose.

Treatment of remaining types:

1. Rest. 2. Keep visitors at a minimum. 3. Do not examine often. Leave the patient alone. If you find by physical signs an area of consolidation there is no necessity for x-rays. 4. Fresh air is important, but do not expose him to the wintry blasts (as was done only a few years back). Oxygen is of great value to combat anoxemia, although patients in delirium sometimes combat the oxygen tent. 5. If there is pain from pleurisy give morphine. If the patient sweats or vomits excessively it is well to remember that the sodium chloride may be decreased.

Conclusion: In any form of treatment be conserv-

ative. If you cannot help the patient do not hurt him. Do not forget antiserums I and II.

HARVARD MEDICAL SOCIETY

The Harvard Medical Society held a regular meeting in the amphitheater of the Peter Bent Brigham Hospital on November 10, 1936.

The medical case was presented by Dr. Richard Lyons: An 87 year old man, who had suffered for 60 years from epigastric pain coming on 3 to 4 hours after meals and relieved by food or soda, entered the hospital for dietary regulation to relieve his increasing pain. His course during this time had been one or remissions and exacerbations. In 1922 an x-ray diagnosis of ulcer was made at the Massachusetts General Hospital; 3 years later he was in the Peter Bent Brigham Hospital for a perineal prostatectomy, at which time a duodenal ulcer was demonstrated by x-ray and a follow-up in the gastrointestinal clinic after discharge was maintained. In 1930 for the first time he began to have coffee-grounds vomitus, with a concomitant fall in the hemoglobin level.

X-ray studies made on the present admission show a peptic ulcer, a left renal calculus, diverticulosis of the large bowel, prostatic calculi and hemoglobin 95 per cent, with a red cell count of five million, and a normal white cell count and differential. Gastric analysis shows a free acid value of 40 points. The hospital course has been uneventful. After unsuccessful attempts to control the epigastric pain with Sippy powders and then with mucin, which the patient disliked, it was found that one gram of bismuth given hourly worked very satisfactorily. Dr. Robert Monroe remarked that ulcer is a benign chronic disease, not susceptible of cure, but commonly well borne for years, as evidenced by this man's tolerance of the ulcer for a period of 60 years with relatively little trouble.

Dr. Emery observed that the case was probably called "hyperacidity" before the days of x-ray, and that the increased frequency with which ulcer is diagnosed today could be explained by the more refined methods of diagnosis. In answer to a question about the occurrence of renal calculi following the ingestion of large amounts of alkali, Dr. Quinby stated that, occasionally, urinary stones were seen after the Sippy régime, but he felt reasonably certain that this man's stone did not arise from such a cause.

Dr. Robert Whitfield presented the surgical case: A 19 year old single male garage helper had fallen from the motorcycle he was riding and struck his right side on an automobile one hour before his admission to the hospital. He was treated in the Outdoor Department for shock; physical examination at that time revealed tenderness and fulness in the right flank, with tenderness in the right costovertebral angle. By x-ray a slight fulness of the right psoas muscle was demonstrated. He was treated conservatively, with the administration of parenteral fluids. For 4 days the urine was grossly bloody, with large clots. On the tenth day profuse urinary hemorrhage recurred, and, after cystoscopy had shown the presence of large clots in the bladder, an immediate right nephrectomy was performed. Intravenous urograms had already shown good dye excretion on the left and none on the right. The postoperative course was uneventful until the seventh day, when the patient complained of pain in the right chest and developed dyspnea, cyanosis, and a fever of 101°. X-rays showed fluid in the right chest and consolidation in the right lung. Several days later pain was felt in the left chest, which was, however, negative to x-ray and physical examination. The wound is now healed, but the temperature continues to rise to 101-103°. Dr. Sosman showed the x-rays, pointing out that in the later plates a broken rib in the right chest had been discovered which could be traced back through the earlier series and which would account for the fluid found in the chest. The pathologist's report on the removed kidney was infarction of the lower half, with hemorrhage, a large clot in the upper part of the kidney pelvis, tears in the serosa, and hemorrhages into the upper part of the kidney. Dr. Quinby explained that nephrectomy had been delayed in this man because he had not presented the usual sign of a ruptured kidney, namely, a palpable tumor in the side formed by extravasated blood and urine. He had sustained a tear of the renal vessels, with subsequent thrombosis leading to two pulmonary embolisms.

Professor Samuel E. Morison of the Department of History of Harvard University presented the paper of the evening under the title "A Few Highlights of Harvard Medical History." The founders of Harvard College wanted law and medicine, as well as the liberal arts, to be taught in the new institution, as, for example, in 1645, when John Eliot made a plea for medical instruction to benefit the Indians and to counteract the practices of the medicine men. President Dunster's request to the General Court for medical books for the library was refused, but the privilege was granted, although advantage was never taken of it, of having once in 4 years a "malefactor's" body for purposes of dissection. In the early days the young medical student associated himself with men already in practice, returning to Harvard, where he took his M.A., to argue a medical question. Competition was naturally keen for the opportunity of working and studying with a celebrated practitioner, and the medical dynasties were already well established. Thirty-five of the 543 alumni of Harvard in the seventeenth century became physicians, while 266 were called to the ministry, certainly a fair number for the young college. Just before the Revolutionary War, Elisha Hersey left the sum of $1,000 to found a chair of medicine. During the Revolution a number of Harvard graduates became distinguished military surgeons, the most notable being John Warren, who was head of the Military Hospital in Boston, and who subsequently founded the Massachusetts Medical Society and made demonstra-

tions in anatomy at the hospital on the site of the present Massachusetts General Hospital. The year 1782 marks the formal beginning of the medical school, for in that year President Willard, Professor Wigglesworth, and Dr. John Warren organized the "Medical Association of Harvard University", which was to grow into the present school. The first three professors, appointed soon afterward, were Dr. Benjamin Waterhouse, a leading practitioner with a European education; Dr. John Warren, only 29 years old at the time, and Dr. Aaron Dexter, just 4 years older. The first lectures were held in Harvard Hall and Holden Chapel. Dr. Warren enthusiastically began making demonstrations immediately, but the others apparently did not begin until 1785. The degree granted for the first few years was that of M.B., until in 1811 it was changed to that of M.D., so that the young graduates might compete on equal terms with those from other institutions. A vexing problem was the one of securing clinical facilities because doctors already in practice were opposed to the idea of allowing students to work in the various hospitals.

The jealousy existing between Dr. Warren and Dr. Waterhouse led to considerable strife in the school, and provided a certain amount of amusement to those who could appreciate it. In 1799 Waterhouse had performed the first vaccinations in the United States, and in 1803 had persuaded Thomas Jefferson to allow its use at Monticello, whereupon Jenner sent him a handsome gold snuffbox, of which he became so insufferably vain that he resorted to a number of tricks to call it to the attention of people.

In spite of the objections of Waterhouse, the medical school was moved to Boston and established on Mason Street as the Massachusetts Medical College, whose only connection with the rest of the university was the granting of degrees. Dr. Waterhouse had, in the meantime, become so intolerable to his colleagues that he was dropped as soon as the first possible occasion arose.

Because of the vicious system of medical education which was firmly entrenched, the school failed to progress from 1840 to 1870, in spite of the illustrious names included in the faculty. The course then consisted in 16 weeks of lectures and demonstrations, with the remainder of the 3 years spent in apprenticeship to a doctor in practice, so that the professors themselves looked on the school not as a training institution in itself, but as scarcely more than a "recruiting ground". The early years of President Eliot's term saw the beginning of great reforms in the school, with the institution of regular tuition and salaries, the expansion of the curriculum to include 3 years of progressive studies, and the establishment of written and entrance examinations. The number of pupils first decreased under the new régime, but very soon increased. Professor H. P. Bowditch of the Department of Physiology became the first full-time professor in the medical school, and experimented most enthusiastically in his little laboratory on the top floor. After a successful public canvass for $200,000, the school moved to Boylston and Exeter Streets near Copley Square in 1886, and 20 years later had expanded so much that it was transferred to the present location. Among the highlights of the later years are the extension of the course to 4 years in 1892, the establishment of the first bacteriologic laboratory in the United States by Dr. Ernst, and the building of the Peter Bent Brigham Hospital in 1912. Because President Eliot wanted to promote research and discovery and to advance the science of preventive medicine as well as to train capable practitioners, in 1908 Dr. Henry Christian became Dr. Eliot's "new type of dean". As a fitting conclusion, the accurate prophecies made in 1783 by Ephraim Eliot concerning the future greatness of the medical school were read and required no comment.

THE OHIO STATE ALUMNI ASSOCIATION OF NEW ENGLAND

The Ohio State Alumni Association of New England met at the Hotel Buckminster in Boston on Friday evening, December 4, 1936. Dr. Robert Zollinger, '27, Associate in Surgery, Harvard Medical School and President of the Association, presided. The speaker of the evening was Dr. Elliott C. Cutler, Moseley Professor of Surgery, Harvard Medical School and Chief of Staff, Peter Bent Brigham Hospital.

Dr. Cutler's topic was "The Future of the State University." He reviewed the beginnings and the phenomenal growth of the state-controlled universities and then pointed out their two great dangers: first, governmental interference and secondly, the difficulties of the almost unlimited and therefore uncontrolled enrollment. The present entering class of Ohio State University is composed of over thirty-four hundred men and women.

In a comparison of the private as opposed to the State University, Dr. Cutler commented upon the failure of the latter to accumulate endowments and wondered whether it was because the danger of governmental control kept away gifts from alumni. He pointed out that certain of the alumni of the great state universities were just as devoted as those of the privately endowed institutions, and that many of them had quite as wealthy alumni who might be prospective donors to their universities. This led Dr. Cutler to a consideration of the future of education in general, and he gave as his opinion that the mounting taxes enforced by the spending of such huge sums by the federal government, certainly would be copied by states and municipalities and would inevitably lead to higher taxation. He pointed out that it was not wrong to view this from a university viewpoint with great apprehension, since when taxes reached a high enough figure they would prevent the accumulation of wealth by individuals and therefore prevent the endowed university from

obtaining further gifts for carrying on their work, and moreover, such a rise in taxation might result in the taxation of the now tax-free charitable and educational institutions.

With their budget partly going to taxes, and their income cut down by the limitation on gifts from rich people, the endowed university would eventually find itself side by side with the state university of today, supported by government taxes on accumulated funds. This hypothetical future would leave education in the hands of the state, and he wondered whether, when that time was reached, the state university would be able to tell the endowed institution how to carry on as an institution where free thinking was permitted and encouraged, even when they had over them the threat of the politician.

Another portion of his address dealt with the possible brakes which could be put upon too great enrollment in the state university today. He pointed out the difficulties in a government-controlled institution of the prevention of enrollment by stiff entrance requirements, of the immediate pruning down of overenrollment by stiffening the curriculum. He felt that the most hopeful method lay in the establishment of the junior college. It might be considered as a two-year division added to the secondary schools of the country, and to such a college could be sent the candidates who did not contemplate higher education in the university schools and thus in the university proper might be grouped only the more serious scholars such as those attending schools for law, engineering, medicine, education, and so forth. Thus the faculty of the university would not be hampered by hordes of young people who somehow or other hope that a degree from a college would give them some wonderful key to a brilliant future.

It seemed clear to all that the state university must struggle with these two great problems: first, the avoidance of interference by the government, and secondly, the erection of some system by which enrollment in universities would be curtailed if the state universities are to serve their highest purpose and be what they should be, centers for the encouragement of thinkers and scholars, untrammeled by any fetters and kept within such bounds that quantity did not destroy quality.

NEW ENGLAND ROENTGEN RAY SOCIETY

The New England Roentgen Ray Society met November 20, 1936, in the Boston Medical Library for a short business session and a program of papers presented by the members of the staff of the Middlesex County Sanatorium.

Dr. Donald S. King discussed two subjects: "Tracheobronchial Tuberculosis with Atelectasis" and "Dangerous Nodose Lesions", both of which he illustrated with a number of cases. When an atelectatic lobe is discovered, the question arises whether it is due to tuberculosis or to something else. The first case was a 44 year old woman with a complaint of hacking cough for 10 months and marked dyspnea for 3 months and a history of having had relief from similar symptoms 10 years before following an operation on her thyroid. The x-ray examination apparently ruled out the thyroid as a cause of the current difficulty, and suggested a diagnosis of carcinoma of the esophagus or of aneurysm. After a bronchoscopy, showing chronic inflammation, had been done, the region was explored by a surgeon, who found a parenchymal lung lesion. The second bronchoscopy yielded a biopsy of tuberculous tissue. In another patient with atelectasis of the right upper lobe and a sputum negative for acid-fast organisms, bronchoscopy made a diagnosis of tuberculosis. A third case was that of a woman who had a sputum positive for tubercle bacilli and whose chest revealed a unilateral wheeze previously called asthma. By x-ray, atelectasis of the left lower lobe was demonstrated, and bronchoscopy showed this to be due to stenosis of the bronchus supplying that area. Dr. Eloesser of San Francisco has classified these stenosing bronchial lesions as intramural, mural, and extramural, according to the position of the causative process, and has also found a diffuse stenosing bronchiolitis which may produce atelectasis. The differential diagnosis involves distinguishing other lesions from suppuration following the lodgment of a foreign body or the growth of a benign tumor leading to atelectasis. The next case was that of a man complaining of pain and hemoptysis, who showed, by x-ray, obstruction to the right middle lobe bronchus, not definitely tuberculous in nature, and whose sputum was negative for acid-fast bacilli. The final case in this series was that of a woman with a minimal left apical lesion, who continued to raise a positive sputum in spite of good collapse by pneumothorax. Bronchoscopy showed tuberculosis of the left main bronchus, and raised a serious question concerning the value of pneumothorax in this instance.

In talking of the dangerous nodose lesions, Dr. King presented first the chest films of a woman who had entered the hospital because of a pelvic disorder suggesting tuberculous salpingitis and whose routine chest plate had shown a small lung lesion. Six months later the disease involved the whole right upper lobe, and within a year had terminated fatally.

A man of 28, negative for tuberculosis clinically and by x-ray in December, 1933, showed a small nodular lung lesion by x-ray in May, 1934, yet presented no clinical evidence of the disease. Four days later he developed a fever and after a week a cavity had formed and was producing a positive sputum.

The next three cases were similar ones, all having nodose lesions breaking out suddenly into active disease after a period of apparent quiescence lasting from 1 month to 4 years. One of these nodose lesions showed the typical "doughnut" form, which is regarded as being of particular prognostic significance,

that is, a small round area of infiltration or density with a center, sometimes scarcely discernible, less opaque than the surrounding process.

The final case of this series was that of a woman of 44, who came to be examined because of "pleurisy". The first x-ray films showed a nodular lesion in the lung interpreted as metastatic carcinoma, but later pictures were very suggestive of miliary tuberculosis.

Dr. Hugh F. Hare spoke on the "X-Ray Diagnosis of Early Tuberculosis", reviewing his experiences at the Middlesex County Sanatorium. Of approximately 15,000 cases studied by that institution, 42 patients whose x-rays were at one time read as negative have subsequently developed tuberculosis. The age distribution of these is significant, as of the 42, 25 were under the age of 20, 14 between 20 and 30, and three between 30 and 35, with none at all older than 35. Thirty-two of the cases, that is, nearly 80 per cent, were in women. Very few individuals who have not had tuberculosis before the age of 30, or at the most 35, contract the disease thereafter, no matter how pronounced the contact. In 5 of these cases the infiltration was located in the apices, in 35 it was subclavicular, and in 2 it was in the mid-lung, bilaterally in both. All of the cases without exception had a history of direct contact. Only 4 had calcified pulmonary lesions demonstrable by x-ray, and 1 case had "scrofula". As an indication of the importance of following with frequent x-rays those in contact with tuberculosis, it was noted that the stage of the disease at the time diagnosis was made ran as follows: Thirty-four were rated as minimal, 5 as moderately advanced, and 3 as far advanced. In 16 cases (40 per cent) constitutional symptoms were present, and in 10 (24 per cent) physical signs could be elicited. In the summary, three points were stressed: First, in the young age group (14 to 25) a negative x-ray gives a false sense of security to those exposed to direct contact with tuberculosis; secondly, this age group requires reexamination by x-ray every 6 months in order to detect minimal lesions, and cannot afford to wait for the appearance of clinical symptoms, and thirdly, pulmonary tuberculosis does not develop after 35 except in patients with diabetes or pneumoconiosis and in the Indian and Negro races.

Dr. Lowrey F. Davenport talked on the "Importance of Blood Studies in the Diagnosis of Pulmonary Tuberculosis", illustrating his points with the graphs kept on several cases. An evaluation of the blood findings and their change from time to time may be very useful in determining whether the tuberculous process diagnosed by x-ray means active disease or a quiescent lesion. The blood sedimentation rate is elevated in the presence of activity, and may show various types of curves in the different stages through which the disease passes. The white count and differential count may give information as to the stage of the process. The beginning tubercle is characterized by an influx of monocytes, which are likewise elevated in the circulating blood. When the tubercle begins to heal, a collar of lymphocytes is formed, with a corresponding increase of these cells in the blood. As a cavity develops, the inevitable secondary infection means a great increase in the number of polymorphonuclear neutrophiles. However, since a patient is rarely in any given stage of the disease, but rather shows all three stages in various locations, the interpretation of the white cell picture offers considerable difficulty. Its greatest value comes in the study of very early lesions with beginning activity, and in nearly healed processes where clinically and by x-ray it is hard to tell whether they are entirely quiescent. It must be realized that there is not always a good correlation between the x-ray findings and the blood studies. To secure the maximum benefit from the blood work, it is desirable to keep a graph of the changes in blood findings, on which is plotted not merely the percentages of each type of cell, but also the total number found. Changes to be noted, which are considered as significant in the direction of healing, are an increase in the number of lymphocytes, associated with healing of the tubercle, a decrease in polymorphonuclears, showing a lessening inflammatory process, a decrease in monocytes, as fewer new tubercles are formed, and a sedimentation rate approaching normal, as the disease quiets down. Since postthoracoplasty cases are hard to study by x-ray, blood work is often of value here. The success of pneumothorax and the progress of reexpansion may be followed closely by repeated blood counts when the films show an apparently inactive process. If the progress of very minimal lesions is followed carefully clinically, by x-ray, and by blood studies, those cases showing good behavior may need no treatment.

The final speaker of the evening was Dr. Harlan Newton, whose subject was "X-Ray Problems in the Surgery of Pulmonary Tuberculosis". Thoracic surgery is entirely dependent upon x-ray, and the procedures of internal pneumolysis and thoracoplasty would be impossible without it. Internal pneumolysis is a common operation because of its great value in increasing the effectiveness of artificial pneumothorax. The pneumothorax must have been instituted at least 3 or 4 months previously to stabilize the mediastinum, make the pleura less sensitive, and lengthen the adhesions. Then with the x-ray one can discover the size of the pneumothorax cavity by the use of stereoscopic films, find a mediastinal hernia if present, and determine the character, attachment, number, and contents of the adhesions. Only the large fibrous adhesions can be visualized, although the surgeon is equally interested in the nonopaque ones. Fluoroscopy helps little in the study of adhesions. Since thoracoplasty today aims to collapse only the diseased lung, the surgeon must know the location of the cavity, whether anterior or posterior, in order to plan a suitable operative procedure. Multiple stage operations are performed to secure the closure of the cavity. If unsatisfactory results are obtained and the disease fails to subside, lipiodol injections should be used to determine whether cavities or

bronchiectasis may be present. Paraffin plombage gives good results in chronic phthisis in the aged, whose disease is not amenable to other varieties of surgical collapse.

During the discussion which followed the papers, Dr. Merrill Sosman commented on his work with the Harvard Medical School students, which showed that about 60 per cent of the men in the first-year class had positive skin tuberculin reactions, while 90 per cent of the fourth-year class gave positive tests, so that 30 men in each class apparently picked up their first infection while in school. None of these first cases have developed clinical tuberculosis as yet, although about one man per class has an activation of a preexisting infection.

Dr. Samuel A. Robins stated that the last patient mentioned by Dr. King in his discussion of nodose lesions had entered the hospital with complaints of a cold, pain in the chest, belching of gas, and constipation. Preliminary diagnosis had been metastatic carcinoma, but the x-ray revealed what appeared to be tuberculosis, and later films showed miliary tuberculosis. However, there developed destruction of ribs, vertebrae, and the neck of the right femur without any soft tissue swellings, and the course had been afebrile, so that the question of miliary metastases to the lung was raised. Dr. Richard Schatzki said that the primary lesion and the present lung pictures appeared to be tuberculosis, while the bone lesions described did not rule out tuberculosis. Dr. Sosman said he wondered whether the patient was sick enough for either miliary tuberculosis or metastatic carcinoma, and asked whether the disease might not be xanthomatosis.

BOSTON UNIVERSITY MEDICAL SOCIETY

The next meeting of the Boston University Medical Society will be held in the Evans Memorial Auditorium, 78 East Concord Street, Boston, on Monday evening, January 18, at 8 o'clock.

PROGRAM

Presentation of Case: "A Physiological Chase of the Circus Elephant," Dr. Francis G. Benedict, Director, Carnegie Nutrition Laboratory.

Medical students, nurses and physicians are cordially invited to attend.

ROBERT E. MOSS, *Secretary.*

NEW ENGLAND PEDIATRIC SOCIETY

FRIDAY, JANUARY 15, 1937

4:00 p. m.

Afternoon Meeting

Children's Hospital Amphitheater

The Use of Ammonium Mandelate in Urinary Infections

Dr. Warren E. Wheeler

Prontosil: Impressions on Its Use in Streptococcus Infections

Dr. Benjamin W. Carey

Presentations of Orthopedic and Surgical Cases

6:00 p. m.

Refreshments at Longwood Towers

6:45 p. m.

Dinner at Longwood Towers

8:00 p. m.

At the Boston Medical Library

Thyroid Disorders in Children

Dr. Henry F. Helmholz, Rochester, Minn.

Discussion by Dr. J. H. Means, Dr. F. B. Talbot, Dr. R. G. Hoskins.

BOSTON MEDICAL HISTORY CLUB

8 Fenway

Monday, January 18, 1937, at 8:15 p. m.

At the Boston Medical Library

"A Hebrew Poem—Dedicated to Elijah Henschel." Harry A. Savitz, M.D.

"Ottmar Rosenbach—Pioneer in the Development of the Concept of Functional Disease and Functional Diagnosis." Hyman Morrison, M.D.

BENJAMIN SPECTOR, M.D., *Secretary.*

BOSTON MEDICAL LIBRARY

The Annual Meeting of the Boston Medical Library will be held in Sprague Hall on Tuesday, January 19, 1937, immediately after adjournment of the special meeting called for 4:30 p. m.

In accordance with the provisions of Article XVI of the By-Laws, the second paragraph of Article XIV of the By-Laws is included in this notice.

By-Laws, Article XIV, second Paragraph: "All resignations shall be made in writing, addressed to the Secretary, before the annual meeting, and any resignation made subsequently to said meeting shall not relieve the member presenting it from his assessment for the current year."

JAMES M. FAULKNER, M.D., *Secretary.*

MASSACHUSETTS GENERAL HOSPITAL

A Clinical Meeting of the Staff of the Massachusetts General Hospital will be held in the Moseley Memorial Building, on Thursday, January 21, 1937, at 8:15 p. m.

PROGRAM

Case Presentations

1. Development in the Public Health Program for the Control of Syphilis. Dr. Joseph E. Moore, Assistant Visiting Physician in Charge of Syphilology, Johns Hopkins Hospital, Baltimore, Maryland.

Discussion will be opened by Drs. Nels Nelson and C. Guy Lane.

Physicians, medical students, nurses and social workers are cordially invited.

Committee on Hospital Meetings

GRANTLEY W. TAYLOR, M.D., *Chairman,*
EARLE M. CHAPMAN, M.D., *Secretary.*

HARVARD MEDICAL SCHOOL

Lecture on "The Care of the Patient," Tuesday, January 19, at 5:00 p. m.—Amphitheater C.

Dr. O. H. Perry Pepper, Professor of Medicine, University of Pennsylvania.

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheater (Shattuck Street Entrance), Tuesday evening, January 26, at 8:15 p. m.

PROGRAM

Presentation of Cases.

Spontaneous Interstitial Emphysema of the Lungs. By Louis Hamman, M.D., Baltimore, Maryland.

Medical students and physicians are cordially invited to attend.

MARSHALL N. FULTON, M.D., *Secretary.*

THE NORFOLK DISTRICT MEDICAL SOCIETY

A regular meeting of the society will be held at the Peter Bent Brigham Hospital Tuesday, January 19, at 8:00 p. m. Tel. LONgwood 5260. Please note that the date has been advanced from the last Tuesday of the month.

Business: 8:00 p. m.

Communications: 8:15 p. m.

Demonstration of an Interesting Patient. Dr. Henry A. Christian.

Adhesive Pericarditis: Some Clinical and Laboratory Aspects of Its Problems. Dr. C. Sidney Burwell.

Contribution from the Department of Roentgenology. Dr. Merrill C. Sosman.

Some Surgical Aspects of Peptic Ulcer. Dr. Robert Zollinger.

Toxic Goiter. Dr. Elliott C. Cutler.

Discussion.

Collation.

FRANK S. CRUICKSHANK, M.D., *Secretary.*

1247 Beacon Street, Brookline.

NEW ENGLAND PHYSICAL THERAPY SOCIETY

The New England Physical Therapy Society will hold its regular meeting on Wednesday evening, January 20, at the Hotel Victoria, 271 Dartmouth Street, Boston. Council meeting at six. Dinner at six-thirty. Program at eight o'clock.

SYMPOSIUM ON TREATMENT BY PHYSICAL THERAPY AND SURGERY OF CHRONIC INTESTINAL TOXEMIAS

a. Physical Therapy—Frederick H. Morse, M.D., Boston; William G. Curtis, M.D., Wollaston; William D. McFee, M.D., Boston.

b. Surgery—Charles W. McClure, M.D., Boston; DeWitt G. Wilcox, M.D., Newton; Edward H. Trowbridge, M.D., Worcester; A. Carleton Potter, M.D., Boston.

General discussion.

All members of the medical profession are cordially invited to attend.

WILLIAM D. MCFEE, M.D., *Secretary.*

41 Bay State Road, Boston.

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, JANUARY 18, 1937

Monday, January 18—

*8 p. m. Boston University Medical Society. Evans Memorial, 78 East Concord Street, Boston.

8:15 p. m. Boston Medical History Club. Boston Medical Library, 8 Fenway, Boston.

Tuesday, January 19—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Treatment of Ambulatory Cases of Diabetes with Protamine Insulin. Dr. Joseph Rosenthal.

11:30 a. m. Massachusetts General Hospital. Eye Nerve Conference. Outpatient Department.

*12 m. South End Medical Club. Headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

4:30 p. m. Boston Medical Library Annual Meeting. Sprague Hall, 8 Fenway, Boston.

5 p. m. Harvard Medical School, Lecture on "The Care of the Patient." Amphitheater C.

Wednesday, January 20—

8 a. m. Massachusetts General Hospital. Grand Rounds—Orthopedic Department.

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. S. J. Thannhauser.

†12 m. Clinicopathological Conference. Children's Hospital Amphitheater.

4 p. m. - 5 p. m. Surgical Pathological Conference. Dr. Cutler and Dr. Wolbach, Peter Bent Brigham Hospital.

*8 p. m. Robert Breck Brigham Hospital, illustrated lecture on "Arthroplasty of the Elbow." Dr. Sumner M. Roberts and Dr. Robert J. Joplin.

*8 p. m. New England Physical Therapy Society. Hotel Victoria, Boston.

Thursday, January 21—

8 a. m. Massachusetts General Hospital. Circulatory Clinic Rounds.

*8:30 - 9:30 a. m. Exchange visit, Surgical and Orthopedic Staffs of the Peter Bent Brigham and the Children's Hospitals, held this week at the Peter Bent Brigham Hospital.

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Transverse Rupture of the Aorta. Dr. W. R. Crowe.

11 a. m. Massachusetts General Hospital. Medical Grand Rounds. Ether Dome.

12 m. Massachusetts General Hospital. Clinicopathological Conference.

*3:30 p. m. Medical Clinic at the Peter Bent Brigham Hospital. Dr. O. H. Perry Pepper.

*8:15 p. m. Massachusetts General Hospital. Clinical Meeting of the Staff. Moseley Memorial Building.

Friday, January 22—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. General Considerations of Coronary Thrombosis. Dr. Cadis Phipps.

12 m. Massachusetts General Hospital. Urological Conference. Outpatient Department.

12 m. Massachusetts General Hospital. Clinical Meeting of the Staff of the Children's Medical Service. Ether Dome.

Saturday, January 23—

*9 a. m. - 10 a. m. Boston Dispensary, 25 Bennet Street, Boston. Hospital Case Presentation. Dr. S. J. Thannhauser.

*10 a. m. - 12 m. Staff Rounds at the Peter Bent Brigham Hospital. Conducted by Dr. Henry A. Christian.

Sunday, January 24—

4 p. m. Free public lecture at the Harvard Medical

School, Building D, Longwood Avenue, Boston. Pain and Its Treatment. Dr. Tracy J. Putnam.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

January 14—Pentucket Association of Physicians. Hotel Bartlett, 95 Main Street, Haverhill, at 8:30 p. m.

January 15—William Harvey Society. See page 41, issue of January 7.

January 15—New England Pediatric Society. See page 85.

January 15—Boston Society for the Advancement of Gastroenterology. See page 1145, issue of December 10.

January 18—Boston University Medical Society. See page 85.

January 18—Boston Medical History Club. See page 85.

January 19—Boston Medical Library, Annual Meeting. See page 85.

January 19—Harvard Medical School, Lecture on "The Care of the Patient." See page 86.

January 19—The South End Medical Club. See page 41, issue of January 7.

January 20—Illustrated Lecture at the Robert B. Brigham Hospital. See page 1256, issue of December 24.

January 20—New England Physical Therapy Society. See page 86.

January 21—Massachusetts General Hospital. Clinical Meeting of the Staff. See page 85.

January 21—Medical Clinic at the Peter Bent Brigham Hospital. See page 78.

January 26—Harvard Medical Society. See page 86.

February 3—American Social Hygiene Association. See page 1186, issue of December 17.

February 25, 26, 27—The New England Hospital Association. Hotel Statler, Boston.

March 6, June 8 and 9—American Board of Obstetrics and Gynecology. See page 78.

March 30 - April 2—First International Conference on Fever Therapy. Postponement notice. See page 52, issue of July 2.

April 21-24—American Society for Experimental Pathology. See page 1075, issue of May 21.

July 6-11—Fifth International Congress of Hospitals. See page 37, issue of January 7.

October 5-8—American Public Health Association Meeting. New York City.

October 25-29—American College of Surgeons. Chicago, Illinois.

DISTRICT MEDICAL SOCIETIES

FRANKLIN DISTRICT MEDICAL SOCIETY

Will meet at the Weldon in Greenfield at 11 a. m. the second Tuesdays of March and May.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

HAMPDEN DISTRICT MEDICAL SOCIETY

January 19—Springfield Academy of Medicine, Springfield, at 4:15 p. m.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

March 16—Danvers State Hospital, Danvers.
May 11—Bear Hill Golf Club, Stoneham.

KENNETH L. MACLACHLAN, M.D., Secretary.
1 Bellevue Avenue, Melrose.

NORFOLK DISTRICT MEDICAL SOCIETY

January 19—See page 86.

February 23—Time, place and details of program to be announced.

March 30—8:15 p. m. New England Deaconess Hospital. A Symposium on Diabetes entitled "A Survey of the Diabetic Work of the George F. Baker Clinic in the New England Deaconess Hospital." Communications and Case Presentations by the Staff. Drs. Elliott P. Joslin, Howard F. Root, Priscilla White, Alexander Marble and Allen P. Joslin.

May—Annual Meeting. Details to be announced.

Note: The Censors will meet for the examination of candidates on the first Thursday of May. Fee of $10.00 is payable at the time of examination. Application blanks may be obtained by writing the Secretary, furnishing name, address and name of school of graduation in medicine. Application must be made at least three weeks prior to date of examination. Candidates whose applications are on file will receive proper notices.

FRANK S. CRUICKSHANK, M.D., Secretary.
1247 Beacon Street, Brookline.

PLYMOUTH DISTRICT MEDICAL SOCIETY

January 21—11 a. m. Bridgewater State Farm.
March 18—11 a. m. Brockton Hospital.
April 15—Annual Meeting. 11 a. m. Ducy Hospital.
May 20—11 a. m. Lakeville State Sanatorium.

FRED F. WEINER, M.D., Secretary.
231 Main Street, Brockton.

SUFFOLK DISTRICT MEDICAL SOCIETY

January 27—Boston Medical Library. 8:15 p. m. Joint Meeting with the Boston Medical Library. "Anthropology." Dr. Carleton S. Coon.

March 31—Boston Medical Library. 8:15 p. m. "Social Insurance—It Affects the Medical Profession." Dr. Charles E. Mongan. Discussion: Dr. Channing Frothingham.

April 28—Annual Meeting. Boston Medical Library. 8:15 p. m. "Problems in Surgical Diagnosis." Dr. Howard M. Clute.

CONRAD WESSELHOEFT, M.D., President,
CHARLES C. LUND, M.D., Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

February 10—Worcester State Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

March 10—The Memorial Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

April 14—Worcester Hahnemann Hospital, Worcester, Mass. 6:15 p. m.: Dinner—complimentary by the hospital. 7:30 p. m.: Business session and scientific program.

May 6—At 4:30 in the rooms of the Worcester Medical Library, Inc., at 34 Elm Street, Worcester, will be held the spring meeting of the Board of Censors.

Wednesday Afternoon and Evening, May 12—Annual Meeting. Time and place for this meeting will be announced in an early spring issue of the Journal.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.

WORCESTER NORTH DISTRICT MEDICAL SOCIETY

January 27—Meeting at the Leominster Hospital, 4:30 p. m.

BOOKS RECEIVED FOR REVIEW

A Health Education Workbook for Teachers, Parents, Nurses, and Social Workers. Kathleen Wilkinson Wootten. 292 pp. New York: A. S. Barnes & Company. $1.50.

Skin Diseases in Children. George M. MacKee and Anthony C. Cipollaro. 345 pp. New York and London: Paul B. Hoeber, Inc. $5.50.

Studies from The Rockefeller Institute for Medical Research. Reprints. Volume 100. 613 pp. New York: The Rockefeller Institute for Medical Research. 1936.

Food and the Principles of Dietetics. Robert Hutchison and V. H. Mottram. Eighth Edition. 634 pp. Baltimore: William Wood & Company. $6.75.

The Medical Clinics of North America. Volume 20, Number 2, September, 1936. St. Louis Number. 685 pp. Philadelphia and London: W. B. Saunders Company.

Bright's Disease and Arterial Hypertension. Willard J. Stone. 352 pp. Philadelphia and London: W. B. Saunders Company. $5.00.

Reports on Chronic Rheumatic Diseases. Being the Annual Report of the British Committee on Chronic Rheumatic Diseases appointed by the Royal College of Physicians. Number 2. Edited by C. W. Buckley. 140 pp. New York: The Macmillan Company. $3.50.

One Hour of Medical History. Benjamin Spector. Volume V. 155 pp. Boston: The Beacon Press, Inc.

Nutritive and Therapeutic Values of the Banana. A Digest of Scientific Literature. 143 pp. Boston: Research Department, United Fruit Company.

Practical Examination of Personality and Behavior Disorders. Adults and Children. Kenneth E. Appel and Edward A. Strecker. 219 pp. New York: The Macmillan Company. $2.00.

Medical Classics. Compiled by Emerson Crosby Kelly. Volume I, Number I, September, 1936. 78 pp. Baltimore: The Williams & Wilkins Company.

Treatment in Psychiatry. Oskar Diethelm. 476 pp. New York: The Macmillan Company. $4.00.

Kompensatorische Nierenhypertrophie. Erik Moberg. 161 pp. Copenhagen: Levin & Munksgaard.

Bakterienfreie Meerschweinchen. Gösta Glimstedt. 295 pp. Copenhagen: Levin & Munksgaard.

The Practice of Medicine. Jonathan Campbell Meakins. 1343 pp. St. Louis: The C. V. Mosby Company. $10.00.

BOOK REVIEWS

The Oxford Medicine. By Various Authors. Edited by Henry A. Christian. 634 pp. New York: Oxford University Press. $10.00.

The feeling that the individual has been neglected by the concentration of attention upon his or her pathology has been increasingly vocalized during the past decade. This volume of six hundred odd pages is another expression of this feeling from the point of view of the psychiatrist, who "has become increasingly interested in the early recognition of psychoses and the possibility of the prevention of their progression into chronic forms". The material is well written. The volume might be more complete if at least one of the authors had been a general practitioner writing for the psychiatrists, or if the control of syphilis had been discussed with some of the detail lavished upon less hopeful aspects. The reviewer is fully aware that this is but one volume of a system of medicine, however, and must not be criticized as an entity. The work is well printed and bound in the loose leaf form. There are the usual number of typographical errors.

Anatomy of the Human Body. Henry Gray. Twenty-Third Edition Thoroughly Revised and Re-Edited by Warren H. Lewis. 1,381 pp. Philadelphia: Lea & Febiger. $10.00.

In this twenty-third and latest American edition of Gray's Anatomy, each section has been brought up to date by incorporating in it new knowledge which has appeared since the twenty-second edition. The sections on Embryology, the Central Nervous System, and the Endocrine Glands have been thoroughly revised; and new material has been added on the physiologic anatomy of various organs. New illustrations have been added and many old ones have been replaced by new drawings, bringing the total to 1,216 engravings, many of them in color. The English B.N.A. terminology has been retained except for the adoption of Langley's nomenclature of the Autonomic Nervous System. New references to the literature have been added at the end of each section.

Of the many excellent standard textbooks of systematic, descriptive anatomy, perhaps that of Gray has best held its own since its first publication in 1858. The luminous original descriptive text of the young anatomist and "perpetual student" at Saint George's Hospital has always constituted its greatest merit, which time has not altered and which successive editions have preserved in its distinctive character. Such a work becomes an accretion in which are embodied not only the personality of the first author but that of successive editors. Dr. Lewis is to be admired and commended for the skill with which, in recent editions, he has perpetuated and enhanced the qualities of this great work.

Applied Physiology. Samson Wright. Sixth Edition. 686 pp. New York: Oxford University Press. $6.00.

This book is particularly welcome to the present reviewer since it is admirably qualified to meet a special need of the class to which he belongs, namely, the large body of clinicians who passed an examination in physiology long ago and, since then, have been increasingly conscious of their failure to follow its subsequent advances. Moreover, as the title indicates, it is not a textbook of pure physiology but rather an interpretation of physiology in its bearings upon questions of clinical medicine.

Thus the first section on the Nervous System begins with a discussion of that important source of diagnostic evidence, the cerebrospinal fluid; and as one reads on throughout the book one feels constantly that he is gaining illumination on clinical phenomena of the most practical sort.

In fact, the clinician may reflect with some satisfaction that much of this new physiology is really the result of clinical research. Yet if the material is thought to include both physiology and clinical medicine the author deserves all the more credit for having correlated and presented it in such a masterly fashion. As is necessary where so much is included in a single volume the discussions are concise, sometimes to a degree which may cause readers to wish for a little more elaboration. On the whole, however, the style is clear and obscure technical terms of the laboratory are successfully avoided, so that every physician may find that it is written in familiar language.

All sections in this new edition are satisfying, especially since they include reference to the most recent work judiciously evaluated; but perhaps most noteworthy are those on the Involuntary Nervous System, the Ductless Glands (including the sex hormones), Metabolism (including vitamins), and the Secretion of Urine. It is a work which may be recommended in the highest terms.